

RESPIRATION

Respiration is divided into FOUR events:

- 1 Pulmonary ventilation inspiration & expiration
- 2 Exchange of gases at lungs: External respiration
- 3 Transport of O_2 & CO_2
- 4 Exchange of gases at tissues: Internal respiration

Non-respiratory functions of the lungs:

- 1 Help venous return by respiratory pump
- 2 Angiotensin I $\xrightarrow{ACE}$ Angiotensin II
- 3 Regulation of acid-base balance i.e CO_2 excretion
- 4 Regulation of H_2O & heat
- 5 Vocalization
- 6 Removal of foreign particles & pathogens

Smoking X • $> 10 \mu$: nose mucus • $< 10 \mu$: lung mucus escalator by cilia
 • Smaller particles : alveolar macrophages

Air passages

- Bronchi repeatedly divide

After 20 generations, the alveoli are reached

- Vagus $\rightarrow$ bronchoconstriction

Symp $\rightarrow$ B_2 receptors $\rightarrow$ bronchodilatation

Alveolar epithelial cells (pneumocytes):

- a Type I : gas exchange
- b Type II : secrete surfactant

VISIT...

LANZAROTE
Caliente.COM

Respiratory zone

- bronchioles, alveol. ducts & alveoli
(muscle)
- No cartilage
- Alveoli i.e gas exchange.
300 million
Area 100 M^2 ملعب قديمي
Thickness 0.2μ .

Conducting zone

(2)

- Dead space
- muscle
- Warming, Humidifying, Filtering insp. air
- Trachea & bronchi
- Cartilage
- No alveoli i.e No exchange.

tert. second. prim.

Mechanism of respiration

Respiratory cycle = inspiration + expiration + pause

inspiration

Active

expiration

Passive

1)

2)

3)

4)

Distension

- 1 mmHg
(Boyle's law)

+

+

-ve

air
in

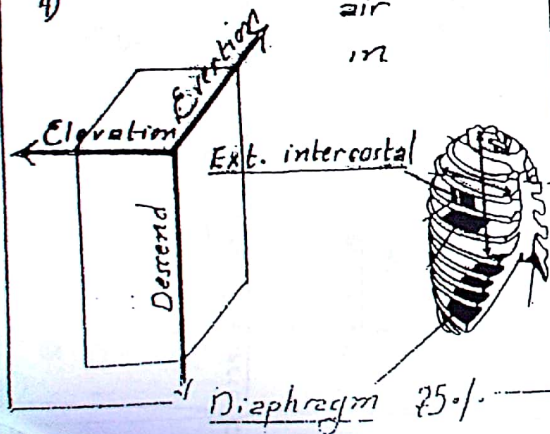
-

-

Recoil

+ve + 1 mmHg
(Boyle's law)

air
out



Accessory insp. muscles

- Sternomastoid
- Serratus anterior
- Scalene

Expiratory muscles

Only with forced expiration

- Internal intercostal ms
- Abdom. wall muscles

R. (3)

2012/08/24

Respiratory pressures

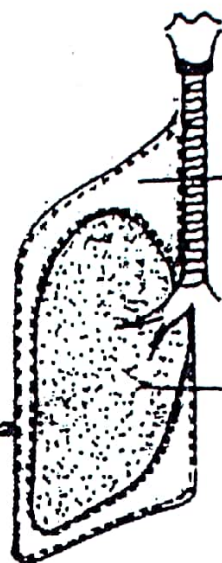
Intrapleural

intra thoracic p

Definition

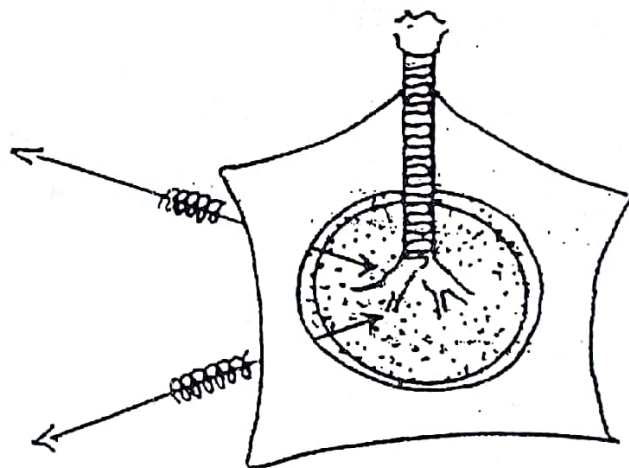
-ve p between the
2 layers of the pleura

Cause



intra pleural
intra thoracic p

intra alveolar
intra pulm p



Continuous tendency

Chest wall to expand

Lungs to recoil

5L

(2.3)

1L

relaxation volume
of chest wall.

end of normal expir

relaxation volume
of lung.

Elasticity

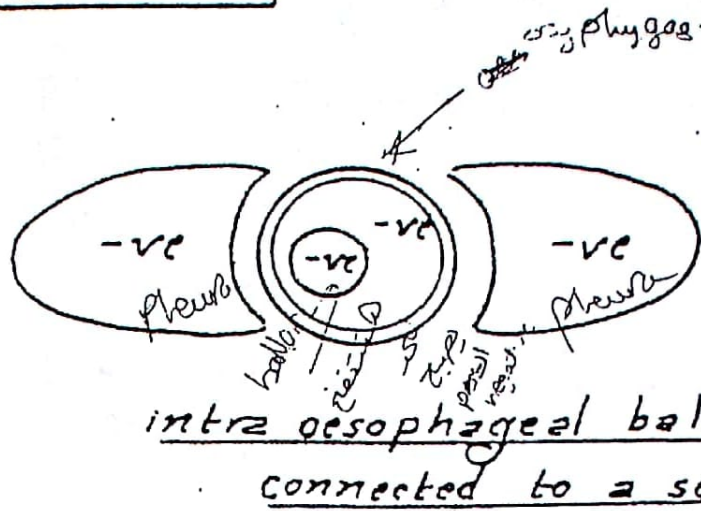
1. Elasticity

1/3 recoil tendency

2. Surface tension

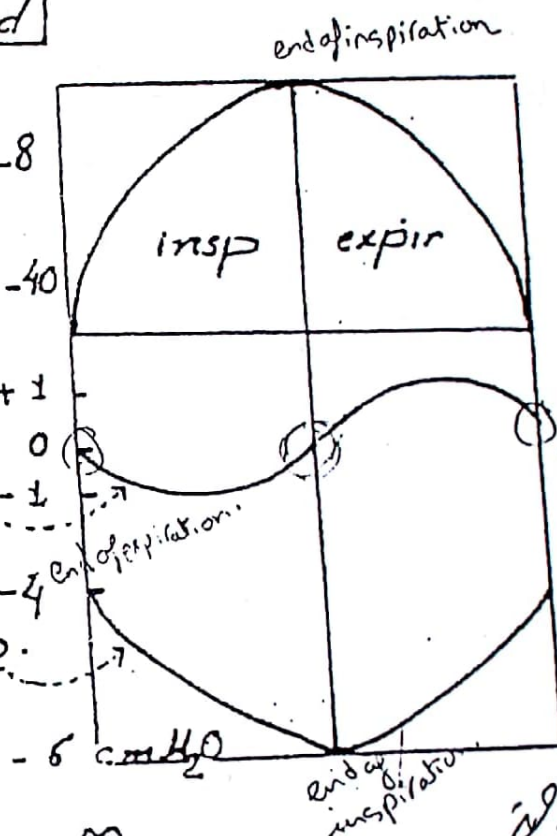
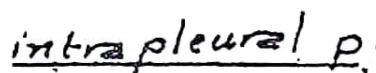
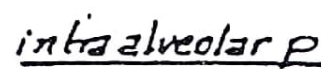
2/3 recoil tendency

(4)

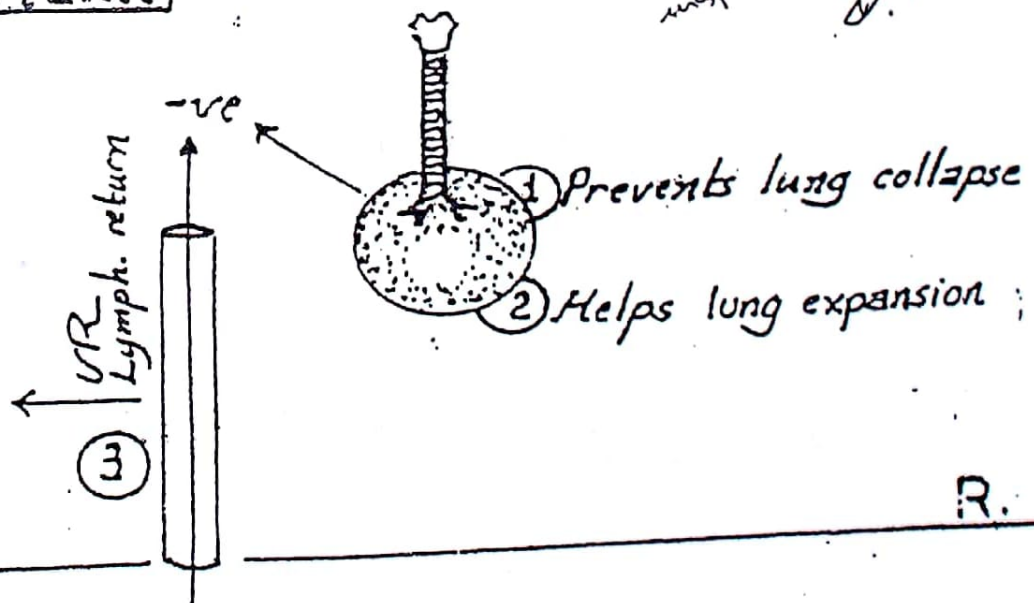
[illegible]

End of normal expir.	-4
End of normal insp.	-6 to -8
Muller's experiment	-30 to -40
<u>Valsalva experiment</u>	<u>+50</u>
Newly born	0 + 1

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End of normal insp.	-6 to -8
Muller's experiment	-30 to -40
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Newly born	0 + 1



The diagram illustrates the relationship between pleural pressure and lung volume. On the left, a vertical cylinder represents the pleural cavity. An upward arrow is labeled $-ve$ (negative), and a downward arrow is labeled VR (volume return). A circled '3' is next to the cylinder. To the right, a lung is shown with a trachea. Two points are marked on the lung's surface: (1) 'Prevents lung collapse' and (2) 'Helps lung expansion'. A circled 'R' is at the bottom right.

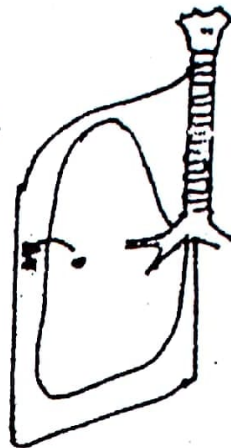


③ Trans pulmonary pressure i.e. transmural pressure

= Alveolar p - Pleural p
Distending pressure of alveoli

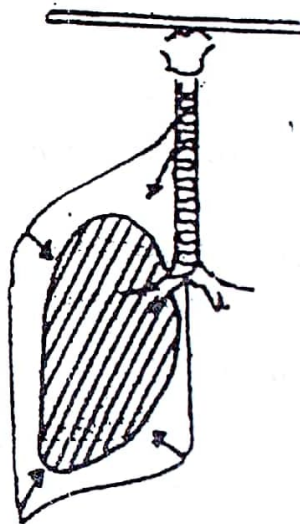
$$= 0 - (-4)$$

$$= 4 \text{ mmHg}$$



Intrapleural pressure becomes positive in:

Ⓐ Valsalva experiment
i.e. forced expiration
against a closed glottis



Ⓑ Pneumothorax

Definition : air in the pleural space

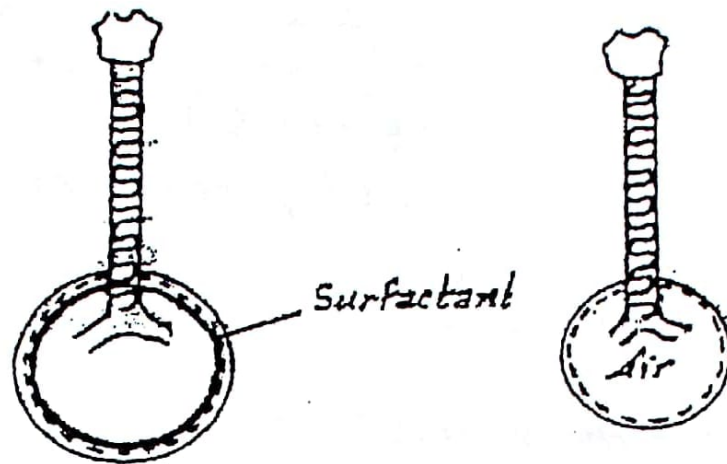
Types :-

- External (open)
- Internal (closed)



Disadvantages : $\neq$ Advantages of -ve intrapleural p

Surfactant



Chemical nature

- Phospholipid dipalmitoyl lecithin
- Surfactant apoprotein
- Calcium ions

Origin

Type II alveolar epithelium
(granular pneumocytes)

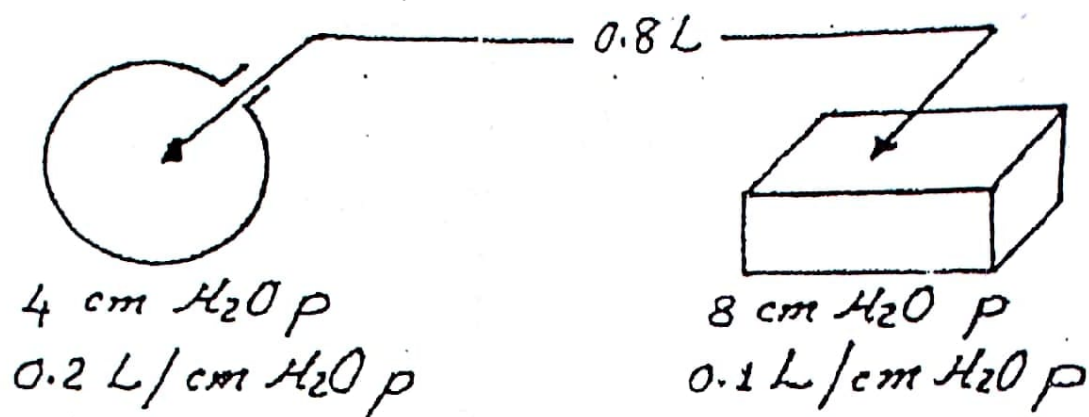
Functions :-

- ① -- surface tension of the fluid lining the alveoli
Apoptn & Ca^{++} allow rapid spread of lecithin
Lecithin forms a lipid hydrophobic surface
 $\frac{1}{2}$ - $\frac{1}{2}$ the surface tension of pure water
- ② Prevents the collapse of small alveoli during expiration
- ③ Prevents pulmonary edema

Surfactant deficiency :-

- ① Respiratory distress syndrome
 - Only in premature baby (--- cortisol)
 - Collapse of alveoli & may death
- ② 100% O_2 for long time in cardiac surgery.
- ③ Occlusion of one pulm. A or major bronchus.
- ④ Cigarette smoking.
- ⑤ Hypothyroidism.
- ⑥ Hypocorticism.
- ⑦ Hyperinsulinism.

Lung compliance



● Definition Ratio $\frac{\Delta V}{\Delta P}$

or change in the volume per unit change in p.
(transmural p or distending pressure
i.e. pressure inside - pressure outside)

● Compliance is a measure of distensibility or expansibility

Compliance is the inverse to elasticity or elastic recoil

● Pressure-volume (compliance) curve of excised lung

The static pressure volume curve (In vitro)

The lung is inflated in 2 ways :-

- By creating a -ve p in a jar around the lung
- By applying a +ve p through the trachea.

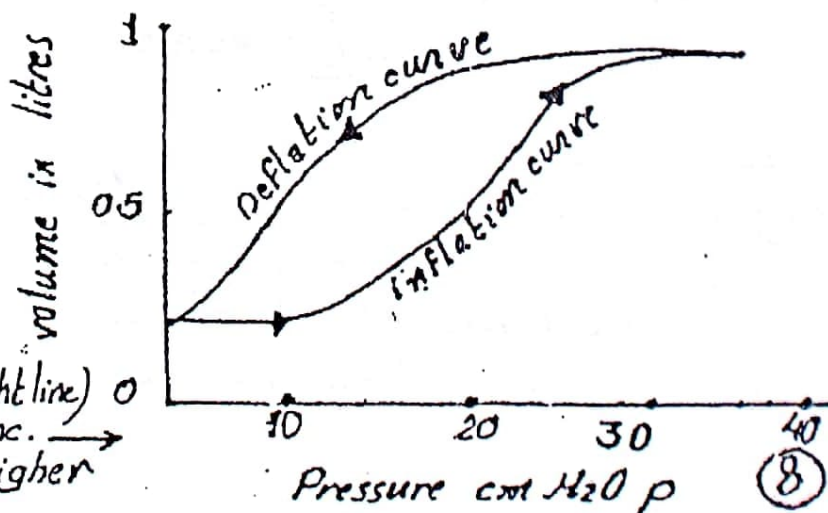
Vol. changes that occur with each change in p. are measured

a Inflation curve

S shaped

→ 10 cm H₂O p
10 - 30 cm H₂O p
30 →

b Deflation curve



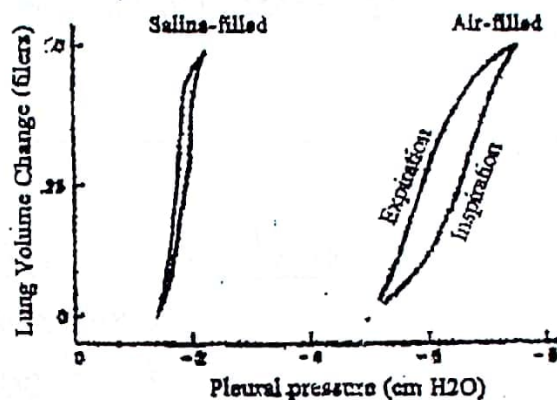
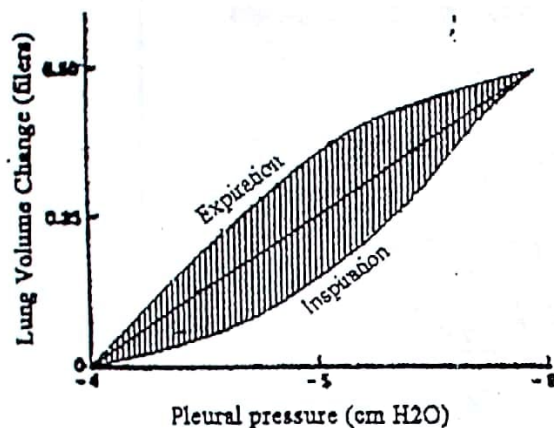
Hysteresis loop (not straight line) 0
in deflation, ++ surfactant conc. →
-- alveolar surface tension → higher compliance

Compliance of the lung in a normal person

The dynamic pressure volume curve. (In vivo)

The transpulmonary pressure is changed in small steps and the lung vol. is measured

$$\begin{aligned}\text{Transpulmonary } p. &= \text{alveolar } p(0) - \text{pleural pressure} \\ &= \text{pleural } p.\end{aligned}$$



Normal standard

- Lungs 200 ml air/cm H₂O p
- Combined lung-thorax system 110 ml air/cm H₂O p

Compliance of the lungs:

- 1) -- in restrictive lung diseases e.g. pulm. congestion & fibrosis and in -- surfactant (RDS) → inspiratory extrawork
- 2) ++ in emphysema & old age → expiratory extrawork

Compliance of thoracic wall:

- 1) -- in poliomyelitis, myositis, arthritis, kyphoscoliosis, obesity
- 2) ++ in athletes

The work of breathing

- The work of inspiration is divided into THREE types:

- ① Compliance (elastic) work most of work
- ② Tissue resistance work
to overcome viscosity of lungs & chest wall
- ③ Airway resistance work $\propto \frac{1}{\text{Total cross sectional area}}$
40% Larynx 40% large airways 20% small airways

- 5% of total energy expenditure $\longrightarrow$ 20% during exercise

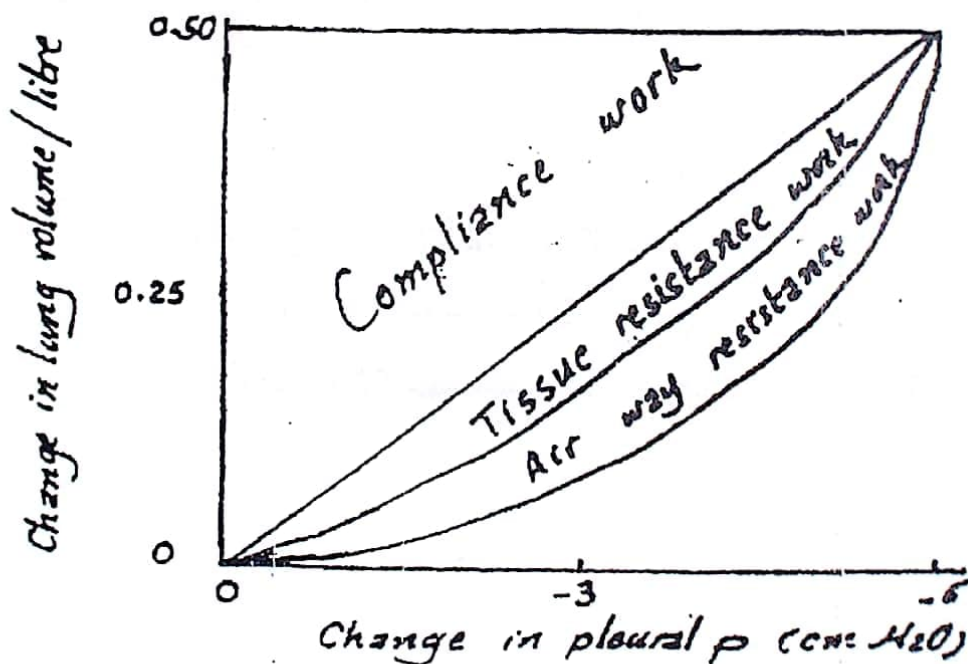
Factors affecting diameter of air passages:

Causes of bronchodilatation:

- 1 Sympathetic (β_2)
- 2 Lung expansion

Causes of bronchoconstriction:

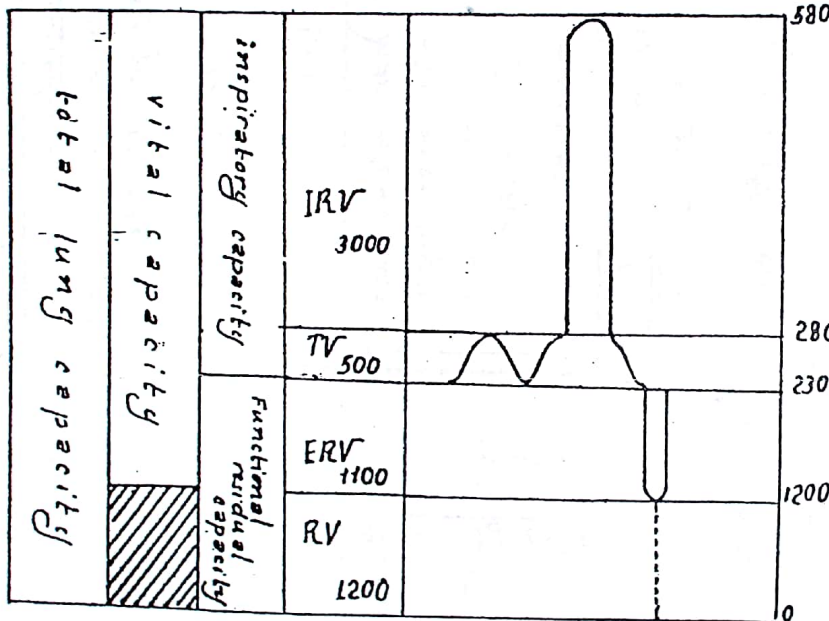
- 1 Vagus
- 2 PCO_2 (direct effect)
- 3 Histamine in allergy.



• The work of breathing is increased when :-

- ① Compliance is reduced
- ② Air passages are narrowed e.g bronchial asthma.
- ③ Surfactant is deficient

Lung volumes and capacities



spirometer

Volumes Capacities

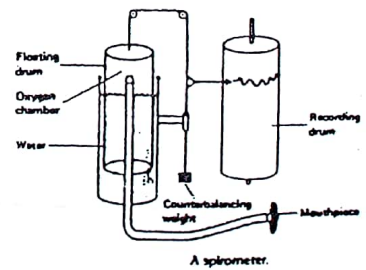
TV (V_T) IC

IRV VC

ERV

dilution method

RV



R.

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Dynamic Lung Volumes

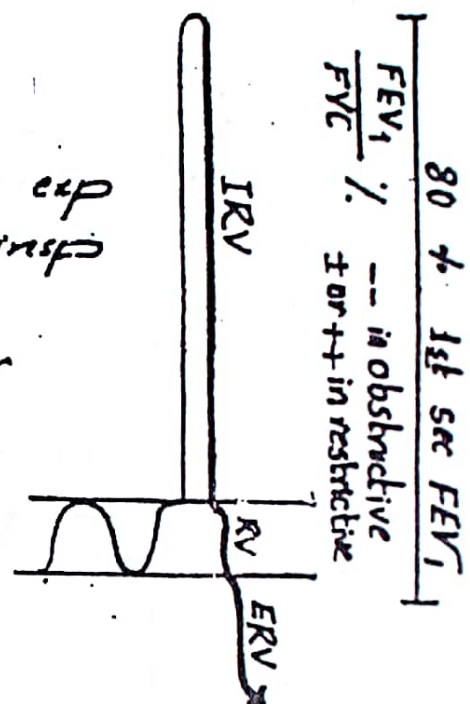
i.e. volumes measured / unit time

- ① Timed vital capacity
- ② Maximal breathing capacity
- ③ Maximal flow rate

Vital capacity:

- Definition max volume exp after max insp
- Measurement Spirometer
- Normal standard

♂	2.5 L / sq. m.
♀	2 L / sq. m.
- Importance: physical fitness
- Factors:

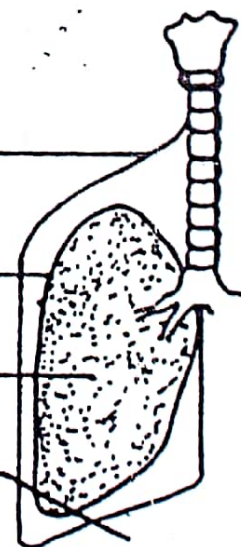


Chest wall
Bone, Muscles

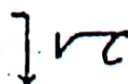
Lung
obstructive
Restrictive

Vol. of blood
if sided Ht failure

Diaphragm
Free descend



Pregnancy & lying position



Maximal breathing capacity = Maximal ventilatory vol. (MVV)

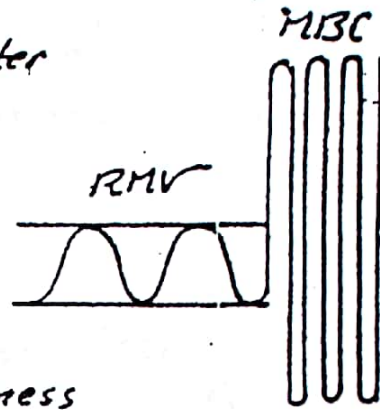
- Definition Max. vol. inspired or expired using fastest & deepest per min.

- Measurement Spirometer

- Normal standard

♂ 80 - 170 L/min.

♀ 60 - 120 L/min



- Importance Physical Fitness

- Factors As vital capacity.

Breathing reserve BR

$$BR = MVC - RMV$$

$\frac{BR}{MVC}$ normally > 90% if < 70% = dyspnea

Maximal flow rate

Maximal velocity of expired air

Peak Flow meter

All Dynamic Lung Volumes are decreased in Obstructive lung diseases

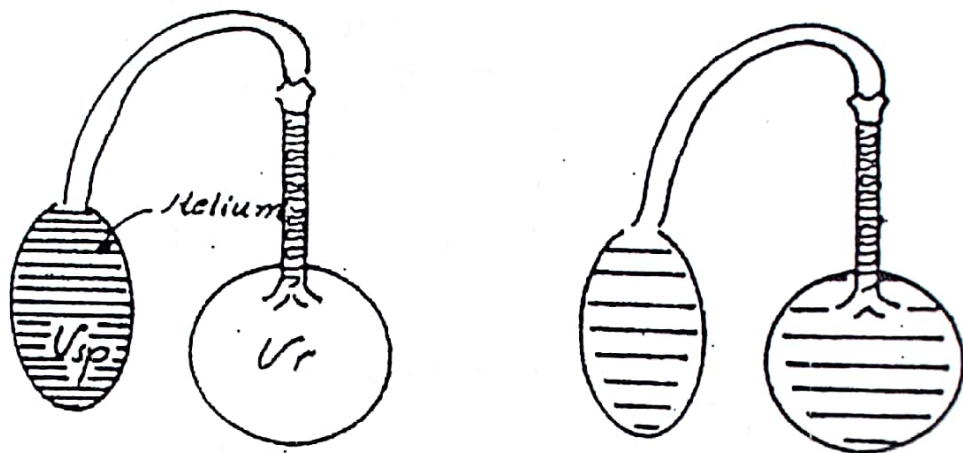
Static lung volumes

$$RV + VC = TLC$$

- ① Vital capacity
- ② Total lung capacity
- ③ Residual volume

Residual volume

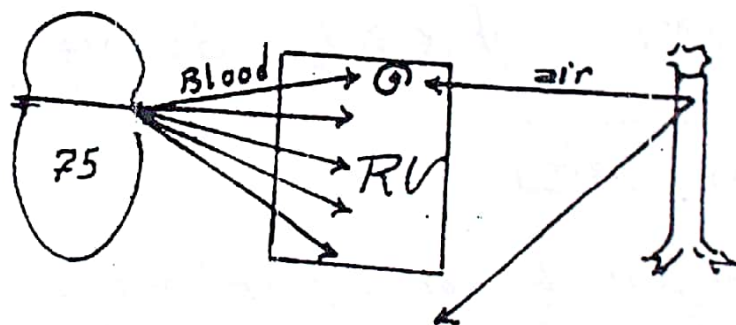
- Definition Volume that remains after forced exp.
- Measurement Dilution principle (Helium)



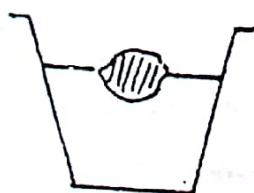
$$C_1 \times V_{sp} = C_2 \times (V_{sp} + V_r)$$

- NS 1200 ml

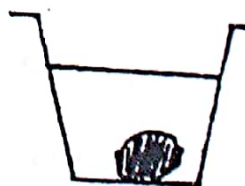
- Importance



- Expulsion pneumothorax
Minimal air



born alive
then killed



born dead

Dead space

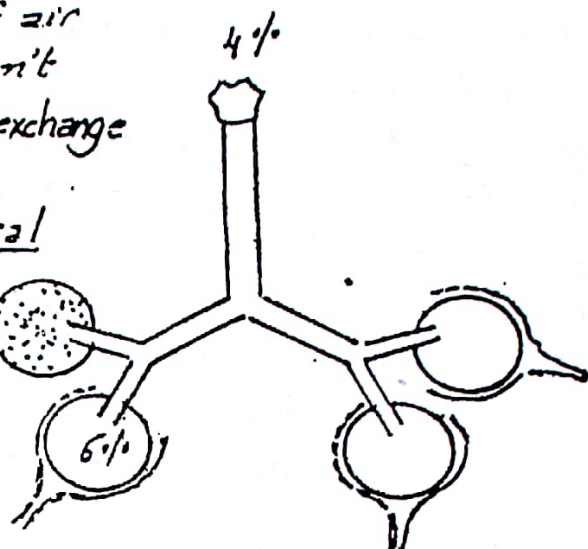
● Definition

the vol. of air that does n't undergo gas exchange

● Types

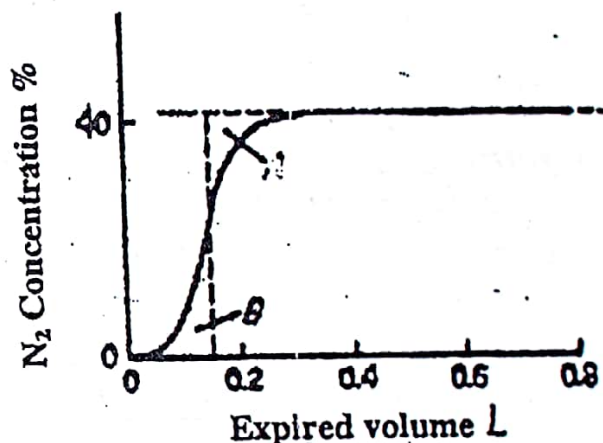
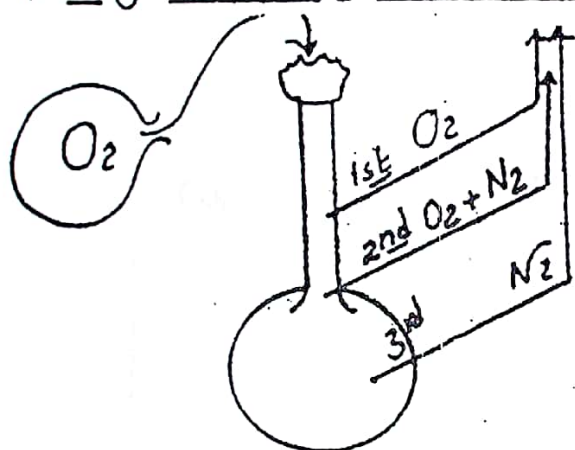
Physiological (total)

Anatomical + Alveolar



● Measurement

1) Single breath N_2 test (Fowler's method): Anatomical DS



2) CO_2 method (Bohr equation): Physiological DS

$$DS = TV \frac{\text{Alveol } CO_2 \% - \text{Exp } CO_2 \%}{\text{Alveol } CO_2 \%}$$

● NS 150 - 166 ml. i.e 30% TV

● Importance

Physiological

warms, fills & moistens inspired air
causes the difference between
alveolar & expired air

Clinical

Shallow rapid breathing
→ Hypoxic Hypoxia

Pulmonary function tests

For working ability and physical fitness

① Pulmonary ventilation tests

a Pulmonary ventilation = minute respiratory vol.
= Resp. rate / minute $\times$ TV = $12 \times 0.5 \text{ L} = 6 \text{ L/min}$

b Alveolar ventilation

= Resp. rate / minute $\times$ (TV - DS)

= $12 \times 500 - 150 = 4.2 \text{ L/min}$

In shallow rapid breathing

Pulmonary vent.: normal Alveolar vent.: decreased

② Static lung volumes

③ Dynamic lung volumes

④ Compliance

⑤ Assessment of regional lung function

Radio isotope technique xenon 133 (insoluble gas)

- Inspired : regional ventilation

- Injected : regional blood flow

Exchange of gases in the lungs

Gas exchange occurs by SIMPLE DIFFUSION

Total p. of the gas mixture = barometric p - H_2O vapour p

Partial p of a gas in a gas mixture

= total p of the gas mixture $\times$ Fraction conc. of that gas

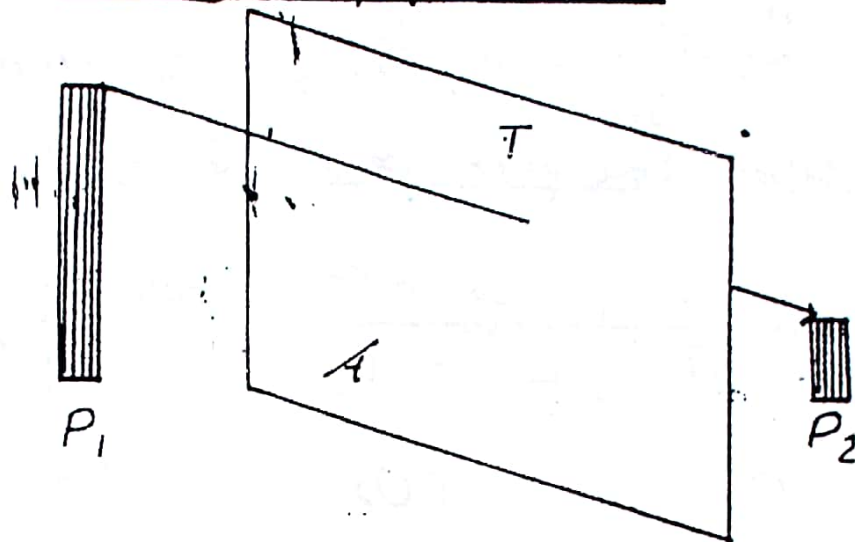
Atmospheric air consists of :-

CO_2	=	0.04 %
O_2	=	20.96 %
N_2	=	79.00 %

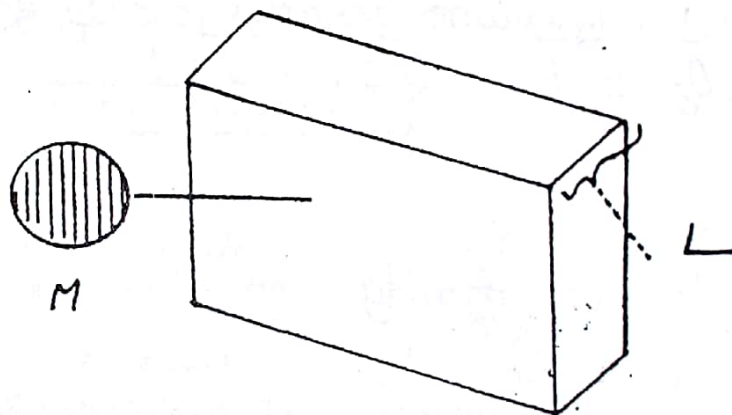
Diffusion of gases

A) In gaseous medium

Directly proportional to :



Inversely proportional to :



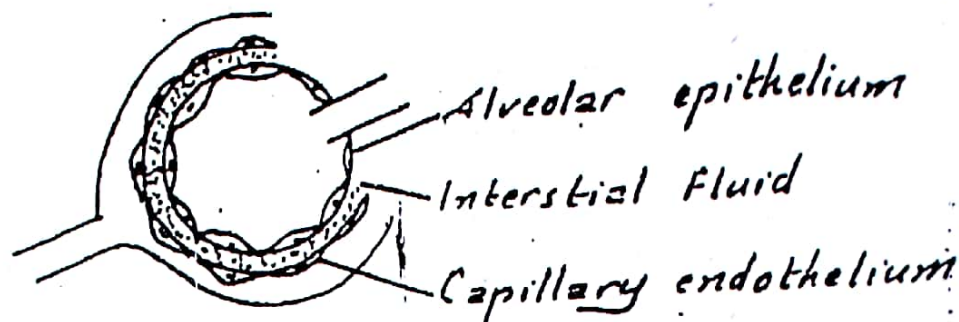
$$DR \propto \frac{P_1 - P_2}{\sqrt{M}} \cdot \frac{A}{L} \cdot T \cdot D$$

	O_2	CO_2
M	1.2	1
$P_1 - P_2$	10	1
	12	1

B)

In aqueous medium

inside the body



$$DR \propto \frac{P_1 - P_2}{\sqrt{M}} \cdot \frac{T}{L} \cdot \frac{A}{D} \cdot \frac{S}{\eta}$$

solubility
viscosity

O₂

CO₂

S

1

24

Solubility coefficient of gas

Gas volume dissolved in 1 ml liquid when gas pressure is 1 atmosphere

O₂ : N₂ : CO₂ = 1 : 0.53 : 20.3

Diffusion capacity

Volume in mL / minute / mmHg

Notes Blood traverses one pulmonary capillary in 0.75 s.
Equilibrium between alveoli & capill. takes 0.25 s

Equilibration of CO₂ takes the time as O₂ inspite of the higher solubility of CO₂ because :

- 1 Pressure gradient for CO₂ < that for O₂
- 2 CO₂ transport in blood as bicarbonate & carbamino compounds, so it takes time to be reversed to free CO₂.

10/12/08/24

Factors affecting diffusion of gases through the pulmonary membrane.

7

• DR is directly proportional to

① $P_1 - P_2$

	Arterial (Alveolar)	Venous (Tissue)	Gradient
O ₂	100	40	60 mmHg
CO ₂	40	46	6 mmHg
		70 mmHg	

② Area

③ Diffusion coefficient

i.e solubility in water

CO₂ : O₂ 24 : 1

④ Temperature

• DR is inversely proportional to

⑤ $\sqrt{A \cdot V}$

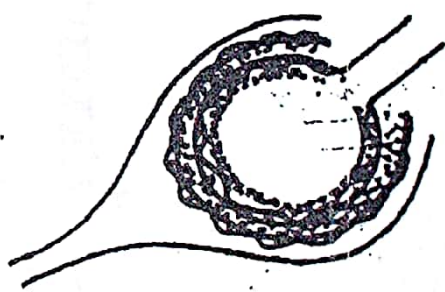
CO₂ : O₂ 1 : 1.2

From 4 & 5 CO₂ : O₂ 20 : 1

⑥ Thickness of the membrane

0.2 μ

6 layers
 Fluid Alveolar epith.
 Basement mem.
 Fluid Capill. endoth.
 Basement mem



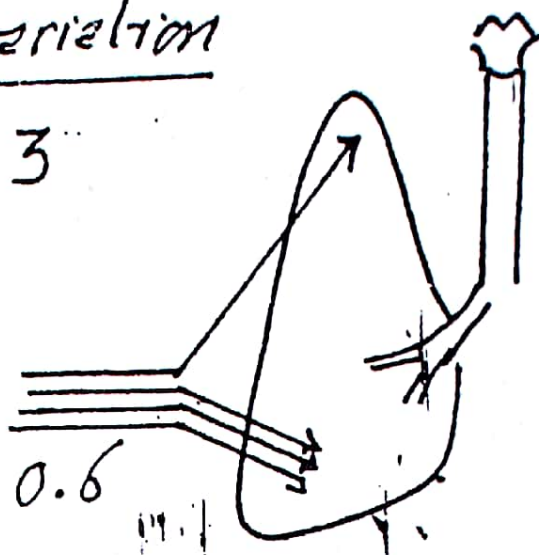
⑦ Alveolar ventilation perfusion ratio VA / Q 9

- Alveolar-vent / COP
- Measurement Radioactive xenon
- Normal standard 4/5 = 0.8

- Physiological variation

Apex $\frac{V}{P}$ 3
↓ P

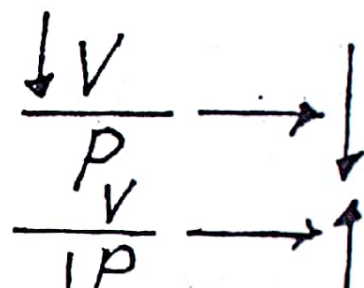
Base $\frac{V}{P}$
↑ P



- Pathological

- Obstructive lung dis.

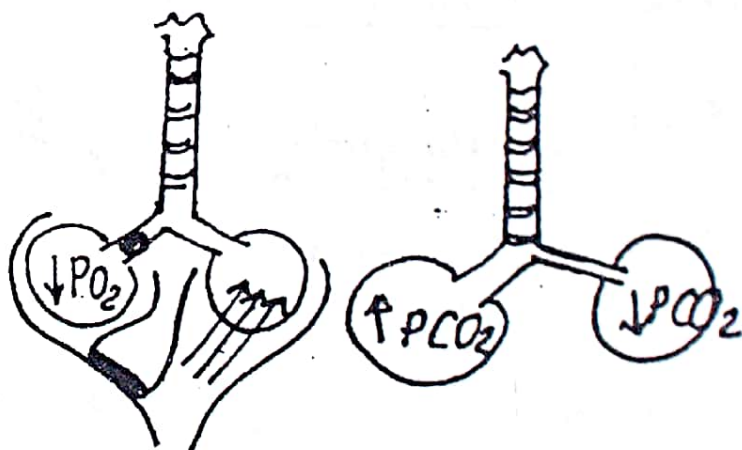
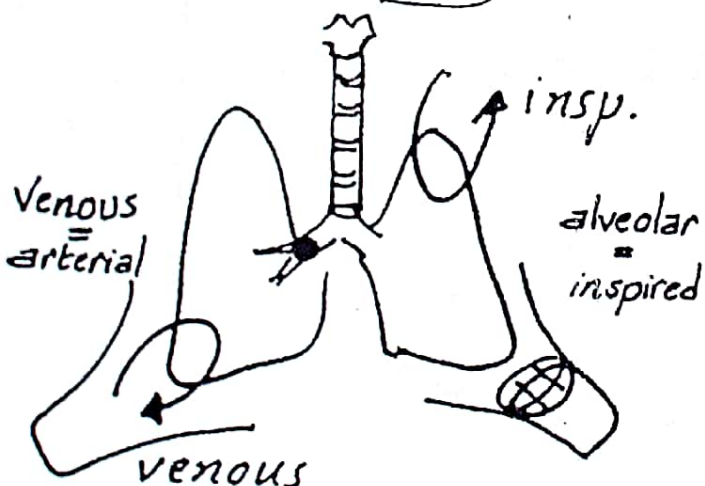
- Pulm. thrombosis



- Apex Less P & Less V

- Matching

- Compensation



- Arterial PO_2 < alveolar PO_2 due to :

1 OxyHb curve is not linear

2 Physiologic venous to arterial shunt from upper airways & coronary venous blood.

O₂ transport

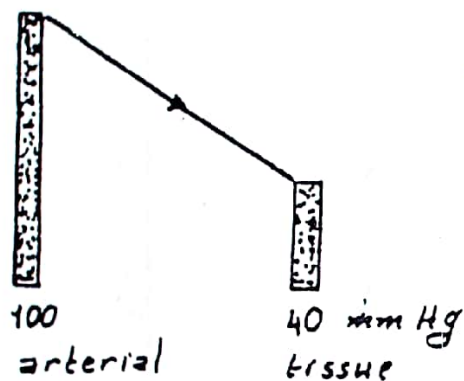
2 Forms

Physical

0.3 ml



O₂ tension

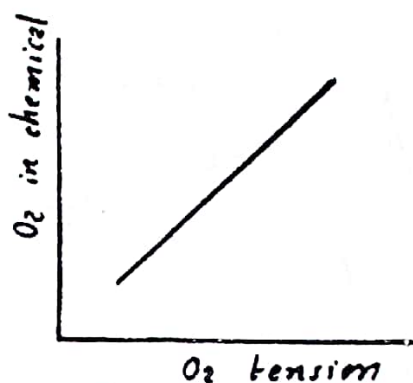


100 cc arterial blood

Chemical

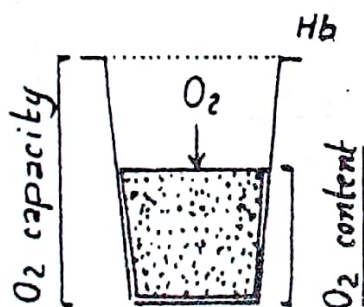
19.5 ml

Hb O₂



Gases in chemical forms have no tension

Definitions



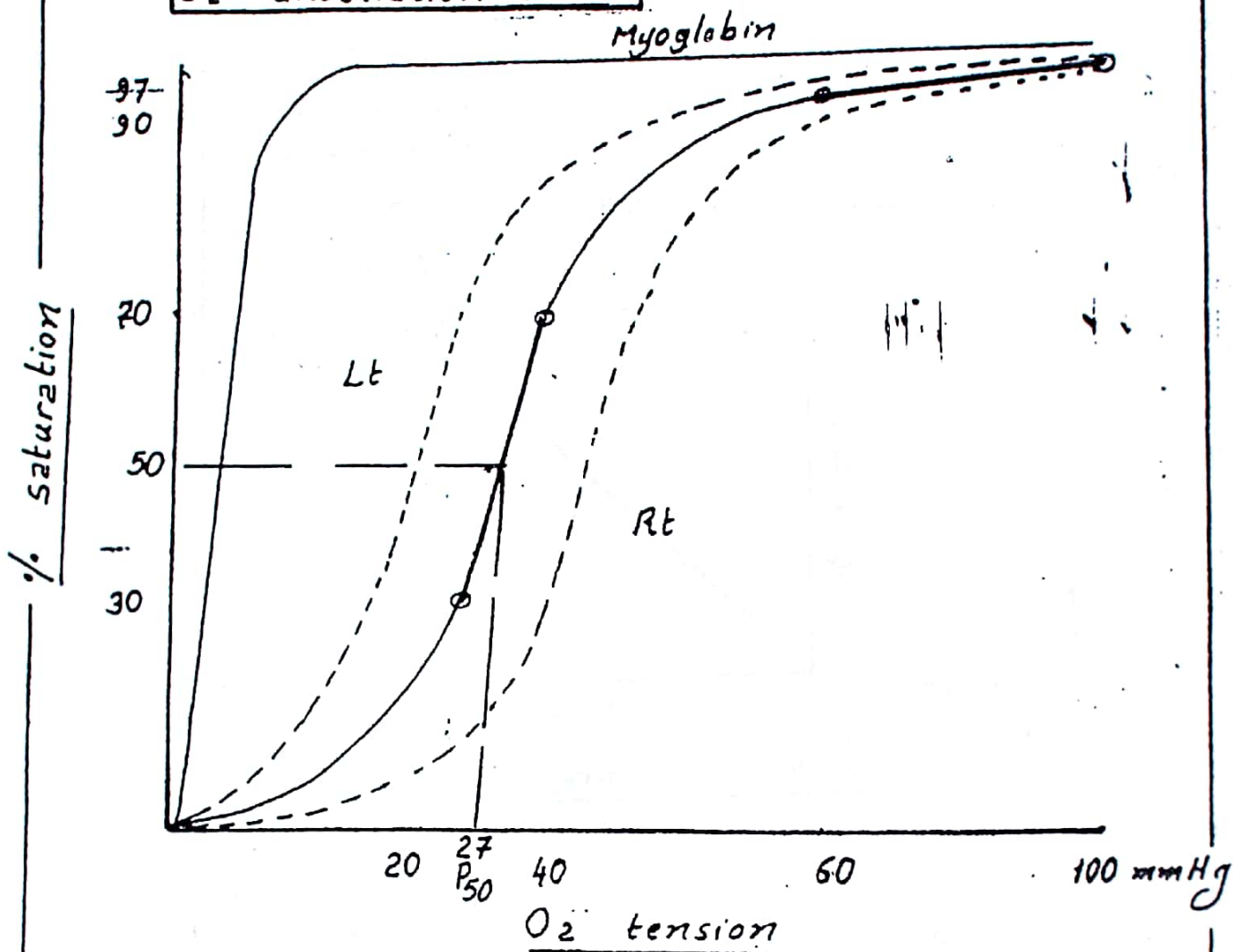
$$\% \text{ saturation} = \frac{\text{O}_2 \text{ content}}{\text{O}_2 \text{ capacity}} \times 100$$

Coefficient of O₂ utilization =

$$\frac{\text{O}_2 \text{ in arterial} - \text{O}_2 \text{ in venous}}{\text{O}_2 \text{ in arterial}} \times 100$$

R.

O₂ dissociation curves



Tissues
Dissociation

Lung
Saturation

Mild exercise

Resting tissues (venous blood)

High altitude

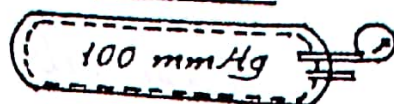
Alveoli (arterial blood)

R.

Method

Tonometer

O₂ tension



O₂ content

19.4

O₂ capacity

20

% saturation

97%

Factors affecting O₂ dissociation curve

	-- affinity unloading Right, ++ P ₅₀	++ affinity of Hb to O ₂ loading of Hb with O ₂ Left (shift), -- P ₅₀
① CO ₂	+	-
② Temp	+	-
③ H ⁺	+	-
④ 2,3 DPG (Glycolysis)	+	-
	Pregnancy Exercise High altitude	CO poisoning

Bohr effect = effect of CO₂ and pH on oxy Hb dissociation curve.

Significance

Hb gives more O₂
at the same O₂ tension

Hb gives less O₂
at the same O₂ tension

% saturation and not O₂ content

↓ Hb
↓ anaemia

↓ O₂ content
↓ O₂ capacity

% saturation
constant

R.
②3

Adult Hb

Fetal Hb

O_2

-- affinity to O_2

++ affinity to O_2

2 Alpha

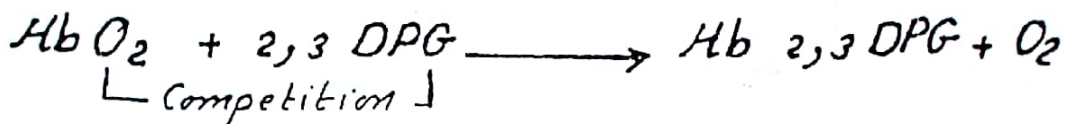
2 Alpha

2 Beta

2 Gamma

can unite with
2,3 DPG

can not unite with
2,3 DPG



Myoglobin

Muscles

Rectangular hyperbola
Can form 1 compound
with O_2

Myoglobin Fe O_2

At rest, fully saturated

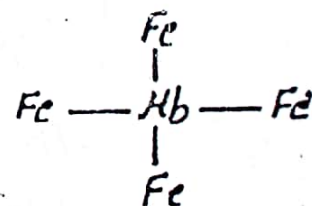
Exercise, it gives its O_2

So, myoglobin acts
as small storage
of O_2

Haemoglobin

Blood

S shaped
Can form 4 compounds
with O_2



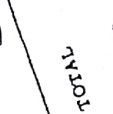
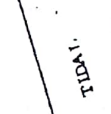
Hb O_2

Hb O_4

Hb O_6

Hb O_8

CO₂ TRANSPORT

		RBC	Plasma	ARTERIAL	VEINS
PHYSICAL		$\text{CO}_2 + \text{H}_2\text{O} \xrightleftharpoons{\text{C.A.}} \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^-$	$\text{H}^+ + \text{HCO}_3^- \rightleftharpoons \text{H}_2\text{CO}_3 \xrightarrow{\text{C.A.}} \text{CO}_2 + \text{H}_2\text{O}$	 42 cc.	 2.6 cc.
CHEMICAL	<u>Bicarbonate</u> <i>More import.</i> <u>Carbamine</u>	$\text{K}^+ \text{HCO}_3^-$ carb HB	$\text{Na}^+ \text{HCO}_3^-$ carbamino ptn	 100 cc.	 1.0 cc.

Arterial Bl. (alveoli)
 Venous Bl. (tissue)

CO₂ tension
 40 mmHg.
 46 mmHg.

O₂ tension
 100 mmHg.
 40 mmHg.

Caused by gases in physical forms

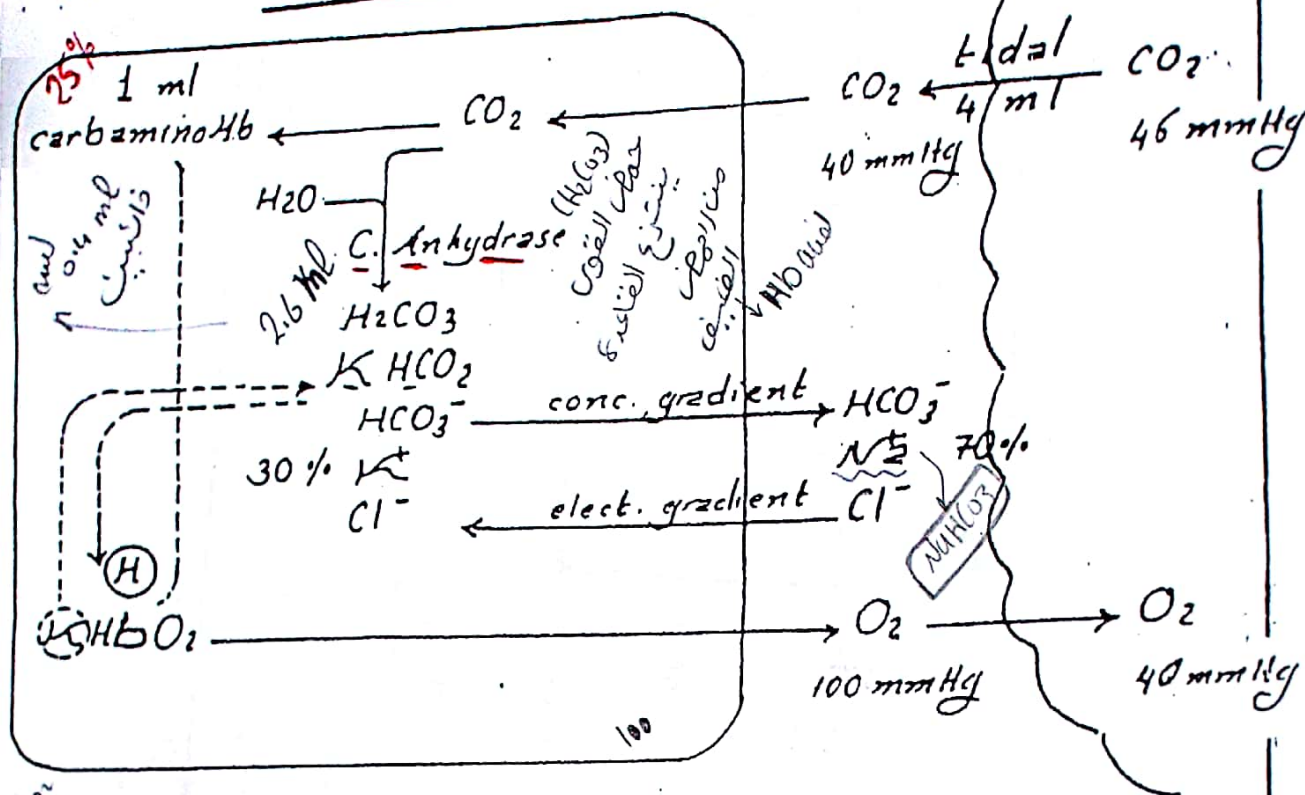
(Buffering of CO_2)

Mechanism of gas exchange at tissue chloride shift phenomenon

RBC

At Tissues
Plasma

Tissues



Donnan's equilibrium $\frac{Cl^- \text{ in plasma}}{Cl^- \text{ in RBCs}} = \frac{HCO_3^- \text{ in RBCs}}{HCO_3^- \text{ in Plasma}}$

Results:-

Items	RBCs	Plasma
HCO_3^-	$\pm$	$+$
Cl^-	$+$	$-$
K ⁺ ions	$\pm$	$\pm$ constant
OP	HCO_3^- Cl^- $\oplus$	$\oplus$
H ₂ O shift	$\leftarrow$	$\rightarrow$
pH	$\oplus$	$\pm$

Reverse occurs at lungs:

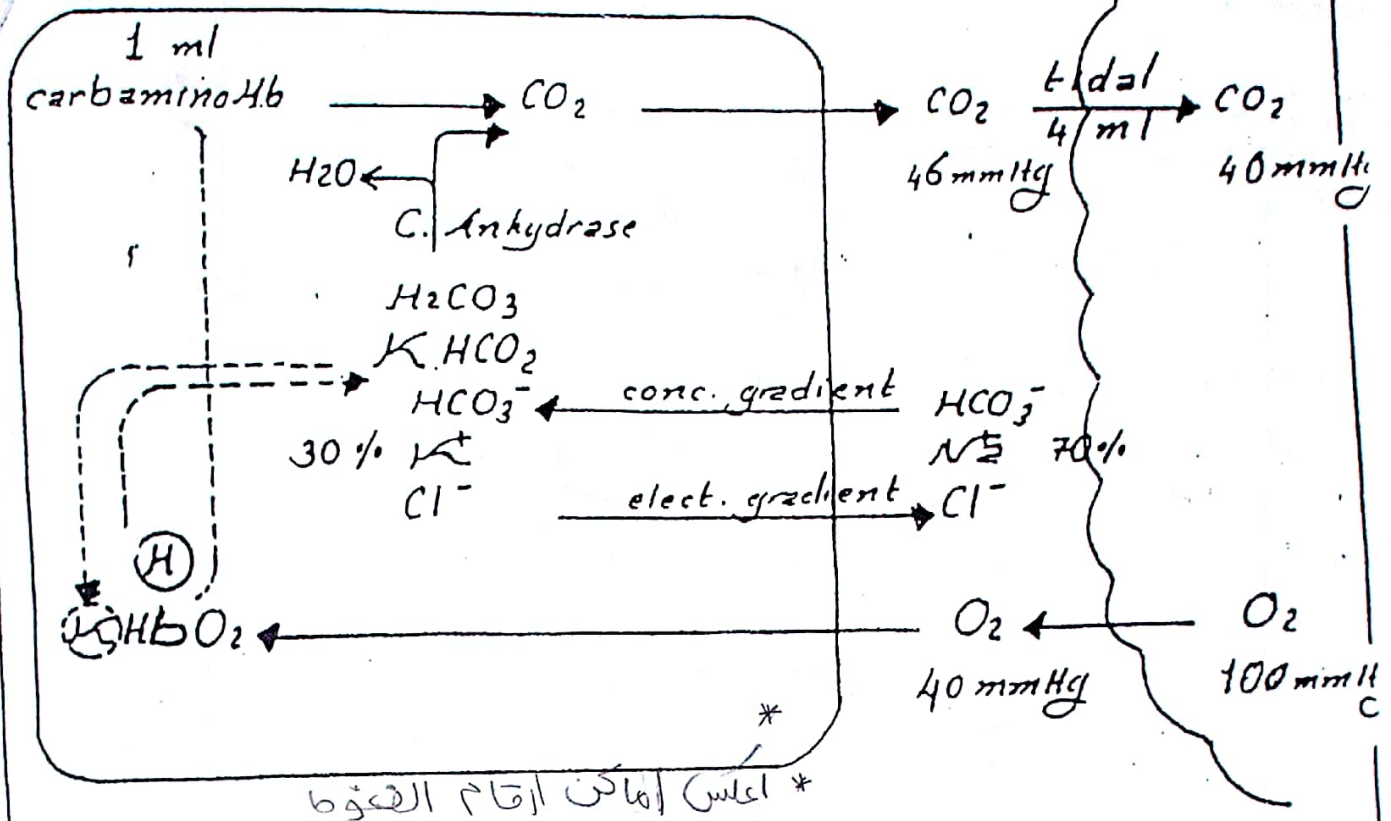
Mechanism of gas exchange at tissues

Chloride shift phenomenon

RBC

At Lungs
Plasma

Alveoli



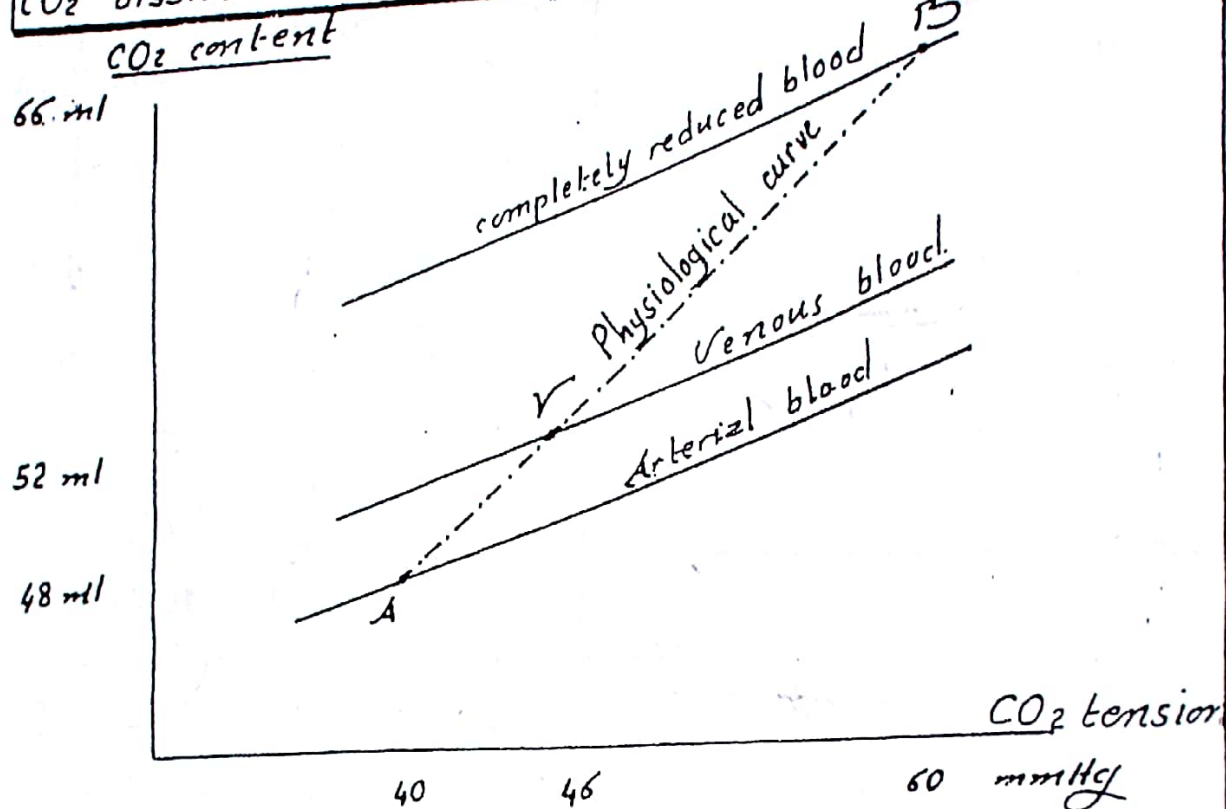
Donnan's equilibrium $\frac{Cl^- \text{ in plasma}}{Cl^- \text{ in RBCs}} = \frac{HCO_3^- \text{ in RBCs}}{HCO_3^- \text{ in Plasma}}$

Results :-

Items	RBCs	Plasma
HCO_3^-	-	-
Cl^-	-	+
Kations	$\pm$	$\pm$ constant
OP	-	$\pm$
H ₂ O shift	→	→
pH	$\pm$	$\pm$

Reverse occurs at tissues

CO₂ dissociation curves



Haldane effect reduced Hb carries more CO₂ & vice versa

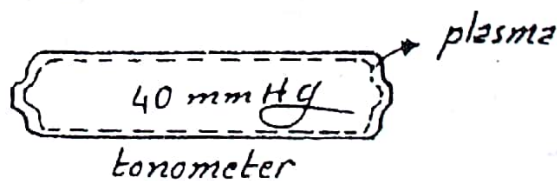
Alk ali reserve

Standard bicarbonate

Definition

Volume of CO₂
in chemical combination
in 100 cc arterial plasma

Measurement



Normal standard

55-70 ml / 100 cc arterial plasma
22-28 mEq / Litre " "

Importance

CO₂ transport
Buffer
Index of acid base balance.

R.

(28)

2012/08/24

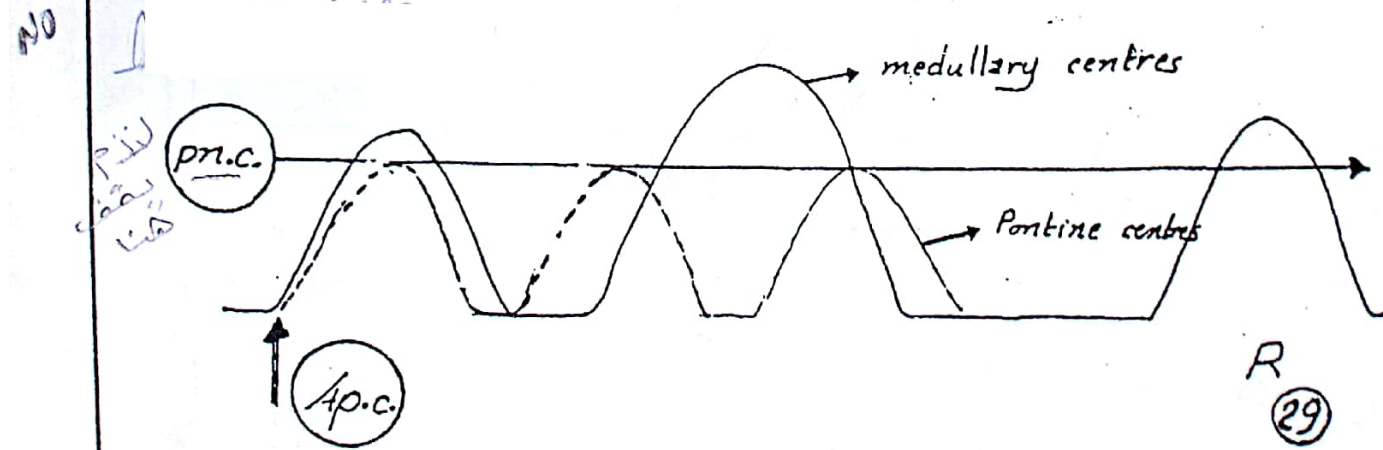
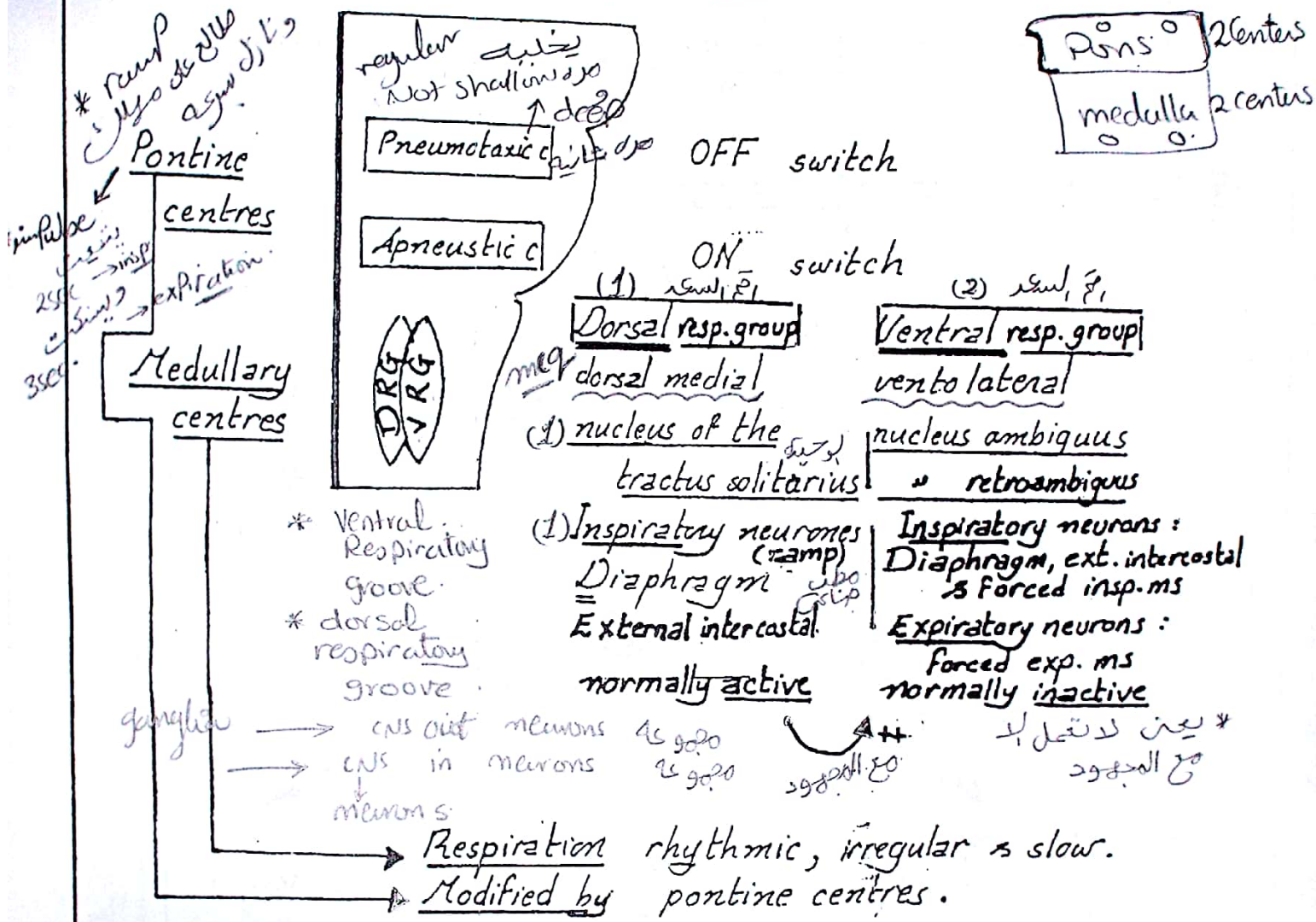
Control of respiration

4 components: ^{receptors} sensors, ^{مراكز} resp. centre, effectors, va feedback ($\uparrow P_{CO_2}$ stim. resp. $\rightarrow \downarrow P_{CO_2}$)

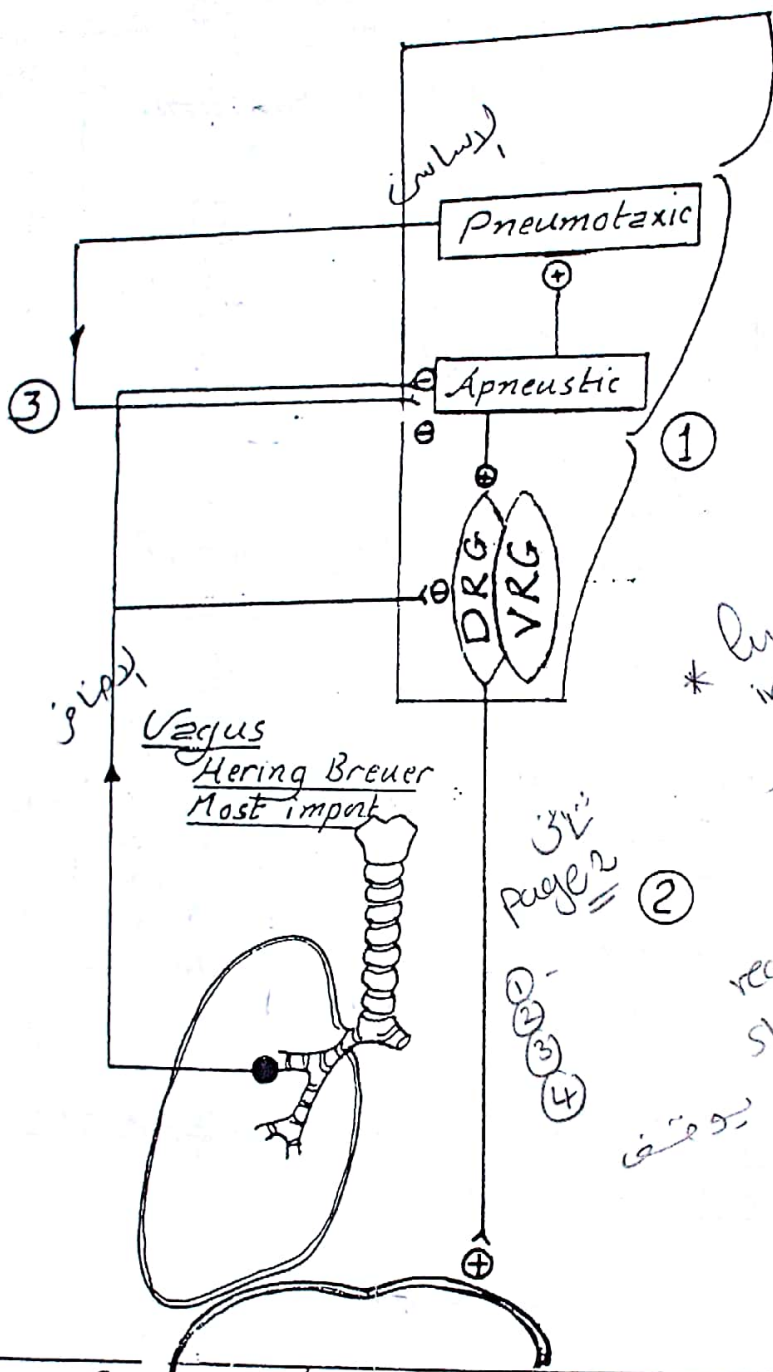
2 pathways:

- 1 Voluntary pathway
Corticospinal tract
Non homeostatic
- 2 Involuntary pathway
Bulbospinal tract
Homeostatic function.

2 regions of respiratory centres:



Genesis of rhythmic respiration



* Lung has receptor in bronchio

لا يتوقف إلا
لأنه يحد من
receptor in bronchio
stretch
و يثبت كسر
الضغط

المرحلة
1 2 3 4

On switch :

المرحلة 1
المرحلة 2
المرحلة 3
المرحلة 4
Apneustic centre
Pneumotaxis

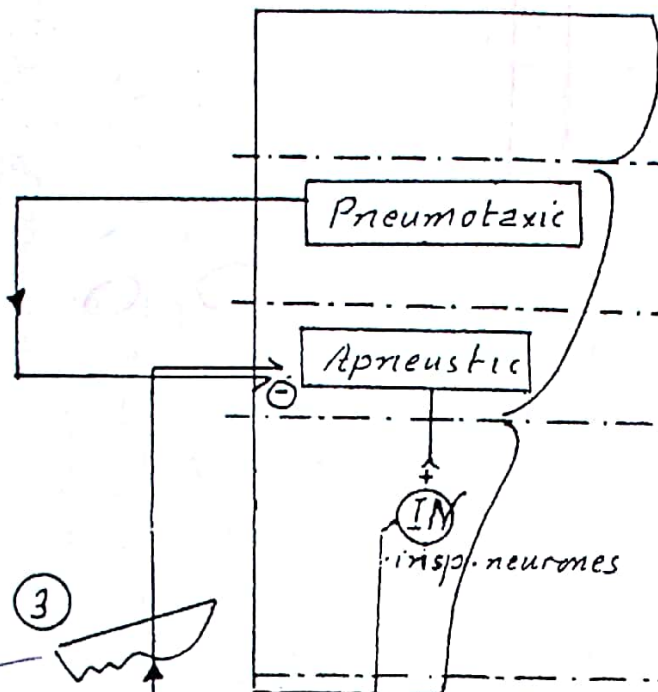
④ Passive expiration
P. 4

OFF switches :-

- Pneumotaxic centre
- Vagus Hering Breuer

Experimental evidence

(Superman)



① TS upper pons
no effect

② TS mid pons
deep & slow

⑤ TS upper medulla
rhythmic
irregular
slow
شعاع
ال
medulla
only

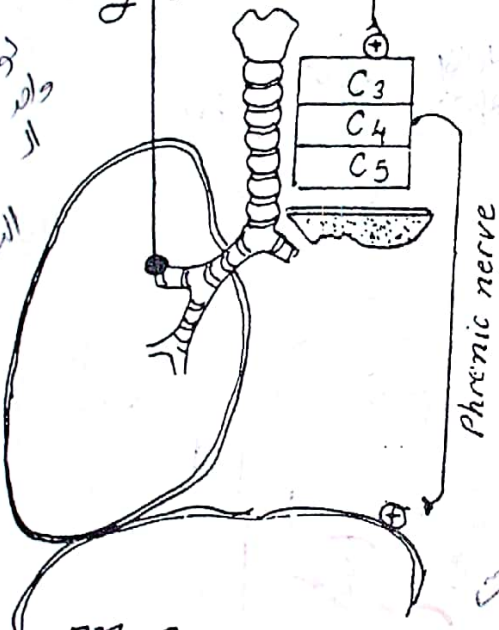
⑥ TS lower medulla
resp Failure
كل مركز التنفس
تأخرت

⑦ TS lower cervical region
diaphragmatic respir.

الكلب في حوض
C7 include
Phrenic nerve from C3, C4, C5
Diaphragm تنفس

④ Apneusis or
Apneustic breathing
كل Both Vagus and
mid pons

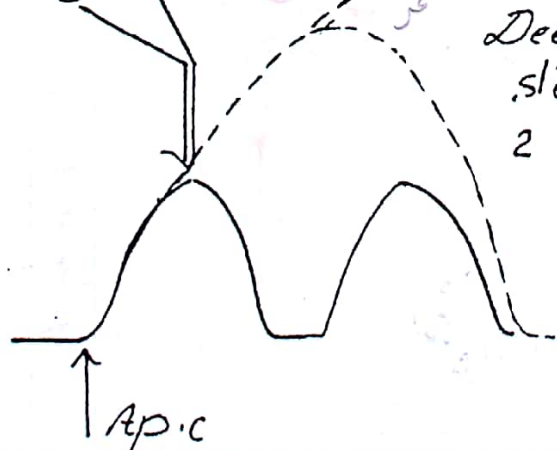
2 8 3
بعد 2 ثواني
الاسترخاء
expirations



pn. c
vagus

شعاع
ال
تنفس

Deep &
slow
2 or 3



5X3

* regulation of respiration
 $\rightarrow P_{CO_2}, H^+, P_{O_2}$

Chemical regulation of respiration

$\uparrow P_{CO_2}, \uparrow H^+, \downarrow P_{O_2}$ stim. resp.

• opposite changes $\rightarrow$ slight inhibitory effect

as tissue work $\uparrow$, $CO_2 \uparrow$, $O_2 \downarrow$
 • Resp. rate $\propto$ metabolic rate

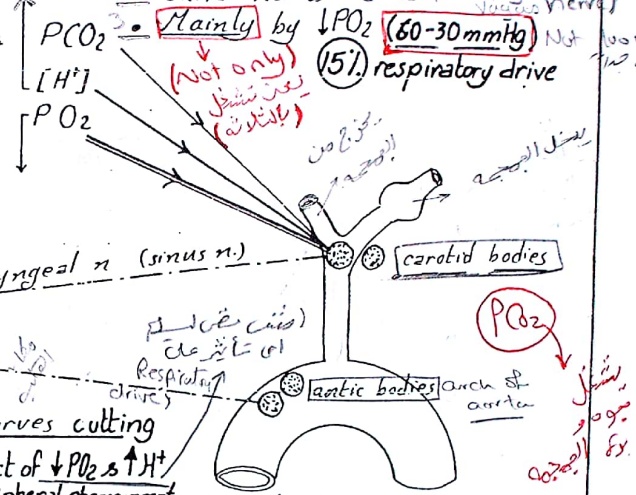
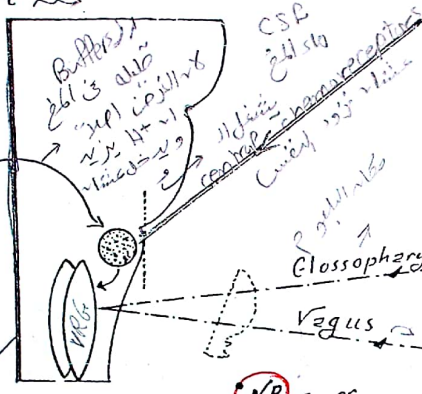
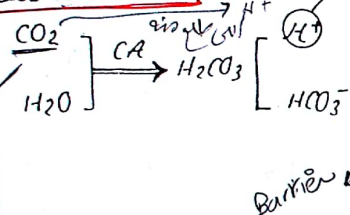
Central chemoreceptors

1. Beneath the ventral surface of the medulla.
2. CO_2 : (85%) respiratory drive
3. Indirect

Peripheral chemoreceptors

1. 2 aortic bodies & 2 carotid bodies
2. Buffer nerves (glossopharyngeal & vagus nerves)
3. Mainly by $\downarrow P_{O_2}$ (60-30 mmHg) (15%) respiratory drive

Less CSF buffers



N.B. Blood H⁺ not cross blood brain barrier

N.B. Buffer nerves cutting

N.B. Narcotics @ G. anaesthesia, only peripheral chemorecept.

0.03% CO ₂	6% CO ₂	10% CO ₂	ventilation 6 times	10 " "	ventilation 10 times	30 mmHg O ₂	ventilation 6 times	20 mmHg O ₂	direct inhibition of resp. centre
-----------------------	--------------------	---------------------	---------------------	--------	----------------------	------------------------	---------------------	------------------------	-----------------------------------

O₂ lack is weaker stimulus for respiration than CO₂ excess because:

1. Hypoxia (100-70 mmHg): stimulatory effect (peripheral chemo), balanced by direct inhibitory effect on respiratory centre.
2. Hypoxia (100-70 mmHg) $\rightarrow$ hyperventilation $\rightarrow$ $\downarrow P_{CO_2}$ & $\downarrow H^+$ $\rightarrow$ slight inhibitory effect.

(32)

Slow

300 Km/hour

intrapleural = intrathoracic

macrophage

300 Km/hour

intrapleural = intrathoracic

macrophage

Reflexes of air passages

Protective reflexes

Stimulus Nerve Response Object (Reflex)

① Sneezing

nose

irritant

Trigeminal (V) (5)

Glottis

Protection of air passages

③ Swallowing

pharynx

IX glossopharyngeal

Glottis

Protection of air passages

② Coughing

trachea

bronchi

larynx

irritant

X vagus

Glottis

Protection of air passages

Pharynx

Glottis

Protection of air passages

34

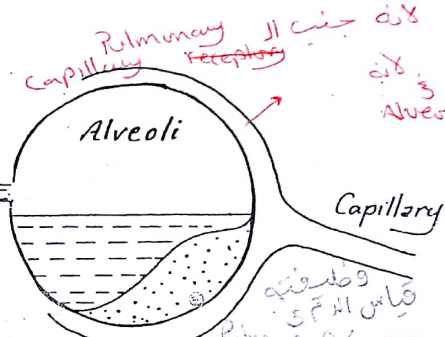
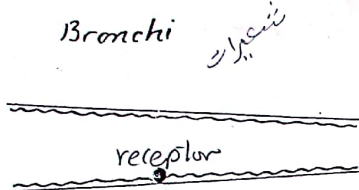
Brower

(tableau)

(Receptors)

Receptors of lungs

inflation + deflation



Site:

Stretch R

(Hering - Breuer reflex)

Lung irritant R

Juxta pulm. capillary R

(J-receptor)

V a g u s

Stretch (inflation)

Inhibitory impulses

Brocho dilatation

(to decrease work expiration)

inhibit ms of bronchi

Slowly adapting R

Irritants

Coughing

Broncho constriction

Apnea

Rapidly adapting R

Pulmonary congestion or edema

Lt sided heart failure

Shallow and

rapid breathing and

(dead space)

* Look Past Page in throat Puffer R.

Co₂ ↑, O₂ ↓

Nervous connection sites: aorta, carotid sinus, aortic arch

(2X4)

(Vascular)

Receptors of cardiovascular system

Atrial (volume) receptors

Low pressure receptors

RA →

Carotid Vagus

* CVP ↑ if VR ↑ Blood volume



vagus



Pressure center of all veins

Harrison's reflex (RAP) → Resp

central venous pressure

* Adrenaline apnea

Arterial baroreceptors

High pressure receptors

Baroreceptors

Buffer nerves

Carotid sinus (IX)

Baroreceptors

vagus (X)

Carotid Vagus



Adrenaline apnea

Resp. VR CO ABP

الضغط في الشرايين الأورطي

Baroreceptors

Pressure

R.

2012/08/24

apnea and any hyperapnea

Cerebral cortex

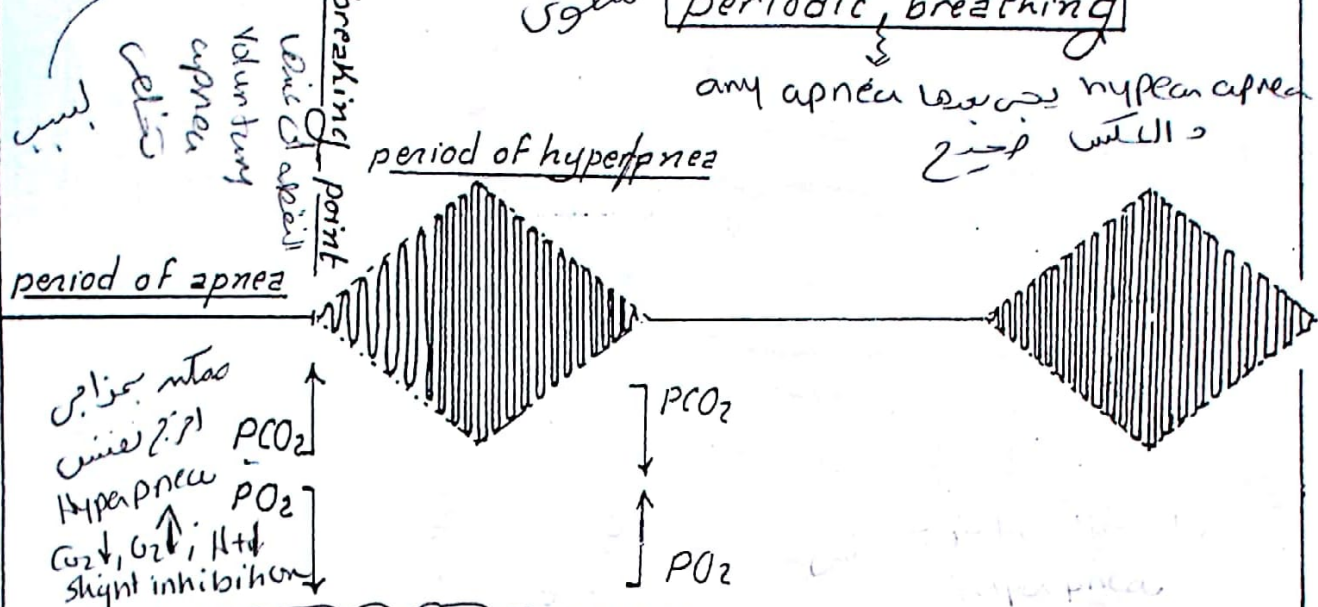
45-60 sec.

شعوى

periodic breathing

(apnea → كتم النفس)

any apnea يجب فيها hyperapnea والنتج فحين



تأثير مجازي
PCO2 ↑
Hyperpnea
PO2 ↓
CO2 ↓, O2 ↑, H+ ↓
slight inhibition

PCO2
PO2

How to prolong apnea?

Hypothalamus

- ① Parasymp. centres → -- respiration e.g. severe pain
- ② Sympathetic centres → ++ respiration e.g. emotions
- ③ Temp. regulating centres → ++ respiration in hot weather
- ④ Panting centres → ++ respiration in hot weather

(الكلب في الكلاب)
الكلب مشع

adrenaline apnea, Swallowing, hyperpnea, Voluntary apnea

Viscera & other reflexes

swallowing
vomiting

Glottis



inhibit
+ apnea
apnea automatic

hiccup

hiccup

phrenic nerve
diaphragm
hiccup

Yawning
Open collapsed alveoli
++ VR



deep insp R.
9 أكم نفس

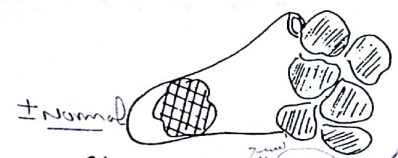
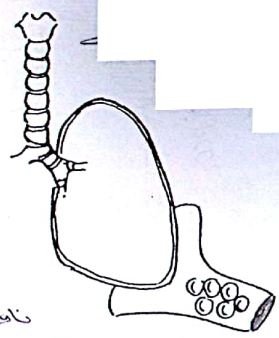
Hypoxic hypoxia -> 1- low P_{O_2}
 2- decrease of P_{O_2}
 3- (more) carbon (poison)

Hypoxia

Definition

O_2 deficiency at tissue level

Types



Hypoxic H

$\downarrow$ O_2 tension of arterial bl.

Anaemic H

$\pm$ O_2 tension
 $\downarrow$ Hb

Stagnant H

$\pm$ O_2 tension
 $\pm$ Hb
 $\downarrow$ BI Flow
 الدم متوقف (أو أبطأ)

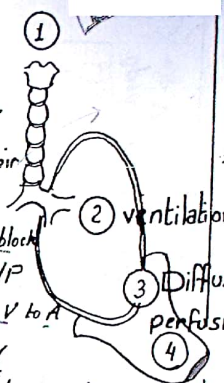
Histotoxic H

$\pm$ O_2 tension
 $\pm$ Hb
 $\pm$ BI Flow
 $\downarrow$ enzymes

Causes

Hypoxic H

- 1 $\downarrow P_{O_2}$ of inspir
- 2 Hypovent
- 3 Alveo-capill. block
- 4 Disturbed V/P
- 5 shunting of V to A



Anaemic H

- quantitative: Anaemia
- qualitative: Met Hb, CO poisoning, Sulph Hb

Stagnant H

- generalised: H. failure
- localised: VC or thrombus

Histotoxic H

- Cytochrome oxidase: Cyanide
- Dehydrogenase: Barbiturate, Alcohol

R.

or beside tissue but can't take it.

Hypoxia and Cyanosis

Cyanosis + C = 4.06 dS

- Content → Not the O₂ carried by Hb
 - Capacity → max. O₂ can be carried by Hb
- Saturation = $\frac{1}{2}$

Normal	Hypoxic H.	Anaemic H.	Stagnant H.	(Histotoxic H.)
Arterial bl. 100 mm Hg 19.4 ml 97% Venous bl. 40 mm Hg 14 ml % saturation	Hb → -- tension ↓ content ↓ saturation ↓	Hb → -- tension (N) content ↓ saturation (N) ± normal relative content capacity So sat. (N)	± tension (N) content (N) sat. (N)	markedly tension (N) content (N) sat. (N)
Cyanosis O₂ under P	present Chemical & physical	no → Physical	present Physical	no → Physical (no improvement)

Cyanide don't

no O₂ in it

5 Symptoms of Hypoxic hypoxia High altitude

It depends on severity, duration, rate and site.

Brain is the most sensitive organ to hypoxia.

● Sudden severe Hypoxia:

-- PO_2 to 20 mmHg $\xrightarrow{15 \text{ sec.}}$ coma 15 seconds
 10,000 m (30,000 feet) $\xrightarrow{\text{death}}$ 5 minutes

● Rapidly developing hypoxia: PO_2 25-40 mmHg (18-25,000 feet)

alcohol intoxications (Mountain sickness)
 - euphoria; slurred speech & drowsiness -- mental judgement
 - headache, nausea.
 - coma & death.

● Slowly developing hypoxia: PO_2 40-60 mmHg (10-18,000 ft) for long time

= symptoms like severe fatigue
 a GIT: Anorexia, nausea and vomiting.
 b CVS: ++ HR, ++ COP and ++ ABP. $PO_2 \downarrow$
 c Resp.: ++ ventilation (stim. of peripheral chemorecep.).

O₂ therapy in different types of hypoxia:

- ① Highly beneficial in: hypoxic H. (altitude, hypovent., --- diffusion)
Anaemic H. (CO poisoning)
- ② Less beneficial in: hypoxic H. (A-V shunt), anemic H., stagnant H.
- ③ Not beneficial in: histotoxic H.

Oxygen toxicity

Oxygen free radicals $\rightarrow$ oxidation of cell membranes & enzymes $\rightarrow$ Destruction of enzymes & nervous tissues

100% O₂ : 8 hours or more $\rightarrow$ irritation of upper passages
 24-48 hours $\rightarrow$ damage of lungs

Retrolental fibroplasia in prematures HMD RDS

Hyperbaric O₂: early O₂ toxicity & CNS symptoms e.g twitches, dizziness, convulsions & coma.

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Cyanosis

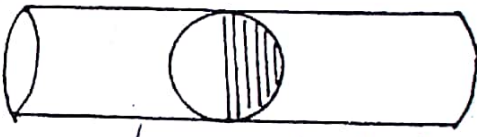
Definition: Bluish coloration of skin and mucous membrane
 ↑ reduced Hb
 Capillaries → thin wall

Threshold: 5 g reduced Hb / 100 cc Capillary Bl.

Types: generalized or localized

Causes: 1 Hypoxia Hypoxic (نقص الأكسجين) Stagnant (مقصور) 2 Asphyxia (اختناق) Hypoxia (نقص الأكسجين)

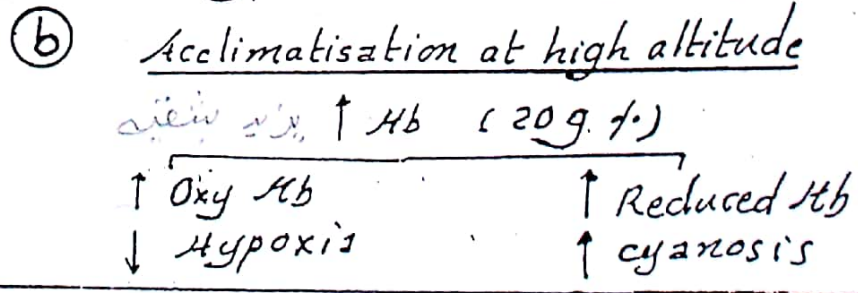
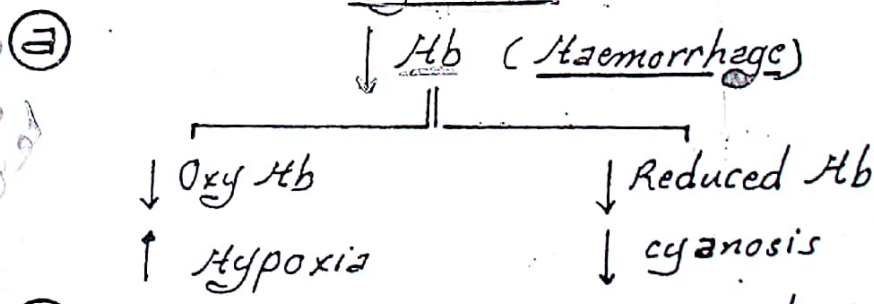
Factors affecting cyanosis: - P_{O_2} ↓ - P_{O_2} ↓, P_{H_2O} ↑, P_{H_2O} ↑
 • Skin thickness
 • Pigmentation
 • Capillary state VC (not severe VC)
 • Blood ← reduced Hb
 ← carboxy Hb
 ← Abnormal composition (lipemia, bilirubin)



Relation between cyanosis and hypoxia: Not parallel

1 In anaemic and histotoxic hypoxia no cyanosis.

2 Hypoxic Hypoxia
 Cyanosis



*cyanosis is not always just color

Handwritten notes on the left margin: "Hypoxic Hypoxia", "Hb", "cyanosis", "not always just color".

Red hypoxia :

A) Cyanide poisoning

15 min

Histotoxic H.

Red in colour

از 1 Hb

oxidizing agent

Met Hb

Ferrous

Ferric

+ cyanide

toxic

→ cyan met HB

non toxic

B) Carbon monoxide poisoning

Anzemic H.

● Production : Incomplete burning of carbon.

● Disadvantages (1) Combines with Hb.

دوبل می شود
Fe²⁺ cell



(2) Affinity ²⁰⁰ 210 times that for O₂.

(3) Shifts O₂ diss curve to left.

● Characters : Cherry red (carboxy Hb).

10-Hb
el 20

Resp. no stimulation

Normal

(O₂ tension is normal).

● Treatment : Remove from the atmosphere
Do artificial respiration : if failure
95% O₂ & 5% CO₂ to stimulate resp.

In sever cases, replacement

Complete rest for several hours (delayed sympt.)
blood transfusion

Acclimatization to low O₂ tension (high altitude)

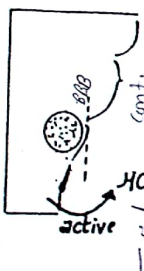
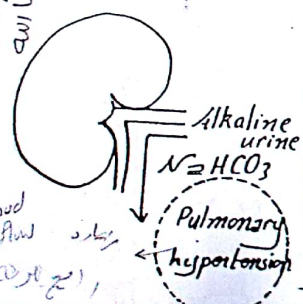
Mechanisms:

↑ O₂
Hyperventilation

↓ PCO₂
Alkalosis

Slight inhibition of resp

2-4 days Stimulation of resp



Pulmonary hypotension

↑ Hb

Hypoxic H

15% 85%
(erythropoietin)
polycythaemia

↑ HR.

↑ Capill.

↑ DPG

Glycolysis
↓ Hb affinity to O₂

anaerobically

↑ Enzymes

Oxidative enzymes
Mitochondria

Hypoxia/hypoxia

- Define
- Causes
- Characters
- Symptoms
- O₂ therapy
- relation bet hypoxic hypoxia & polycythaemia

Ziwi

80% cyanosis R.

2012/08/24

- acclimatization to low O₂ tension (high altitude) -

نظري فقط

(B)

Problems of increased barometric pressure

I Descent = compression = $\uparrow P$

$\uparrow P \rightarrow \uparrow$ dissolved gases

• $\uparrow CO_2 \rightarrow$ no effect $\rightarrow$ لا تأثير

• $\uparrow O_2 > 12h \rightarrow O_2$ toxicity $\rightarrow$ سمية الأكسجين

• $\uparrow N_2$
1st

euphoria

then

then narcosis

10 meters

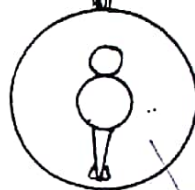
Atmospheric p.

1
2

Solution: replace N_2 by helium

حل: استبدال النيتروجين بالهيليوم
لأن الهيليوم لا يذوب في الدم
بسهولة مثل النيتروجين

50 meters



6

II Ascent = decompression $\rightarrow$ $\downarrow P$

Slow decompression

excess N_2 expired

Rapid decompression leads to:

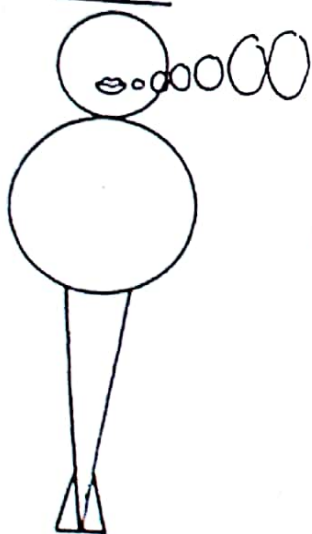
A Decompression sickness (caisson's disease)

excess N_2

Slow

Decompression

Rapid



N_2 in body will go out
so no problem

Bubbles

- Tissues
- Bl vessels



Port 1
N₂ in body
will go out
Bubbles

Treatment

Slow

recompression, then decompression

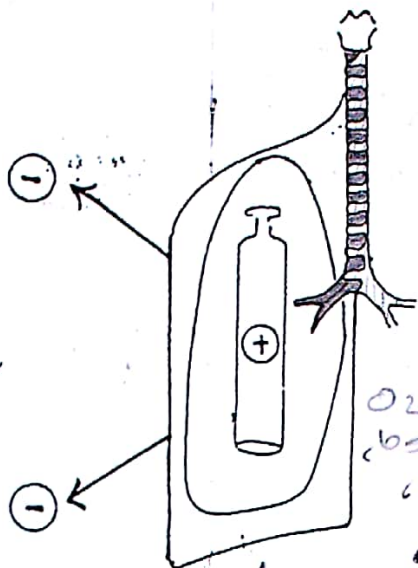
44

⑧ Air embolism

SCUBA

IF the diver

- Holds his breath
- Rises rapidly to the surface



داحضات باء الضغط كاس
على مدرج والاسطوانة من
الضغط - يخرج مدرج من
الضغط (لذا ما اخذ الاز
الضغط من داخل حدة
+ So alveoli will rupture
Pulmonary cap -> rupture

Effect of muscular exercise on respiration:

++ pulmonary ventilation

Mechanisms:

I Metabolic

Mild to moderate exercise: No change in PO_2 , PCO_2 , H^+
Severe exercise: ++ PCO_2 , ++ H^+ , -- PO_2

II Neurogenic (Werge im Pulse)

1 Cerebral cortex (resp before exercise) & hypothalamus (body temp)

2 Proprioceptors

3 stimulate atrial receptors (Harrison's reflex)

4 ↑ VR

5 ++ catecholamines

6 ++ body temperature

Dyspnea

Definition

Consciousness and discomfortable resp. movements

Dyspnoeic index

$$= \frac{BIR}{MBC} \times 100 = 90\%$$

Max. Breathing capacity IF less than 70% So, dyspnea.

45

Breathing reserve

120 - 6 = 114

(tripled)

less than 70

80 - 170 L/min

60 - 120 L/min

80 - 170 L/min

60 - 120 L/min

80 - 170 L/min

60 - 120 L/min

Causes of dyspnea

④ Neurogenic:
(Emotional)

حالة نفسية

↓
O₂

③ General:

Acidosis H⁺ ↑
↓ O₂ ↑ CO₂

* chemical regulation of respiration

① Respiratory:

a) Ventilatory

- Obstructive
- Restrictive

b) Diffusion defect

Fibrosis > 0.14m

c) Perfusion defect

② Cardiac:

Lt ventricular failure

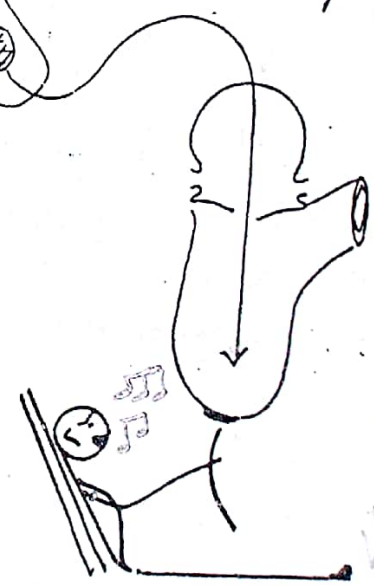
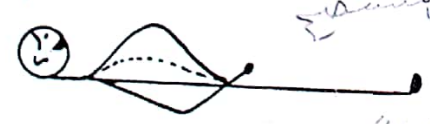
Pulmonary congestion

stimuli (rapidly adapting R) receptors
-- vital capacity

⑤ ++ metabolism

Hypert thyroidism, fever

Orthopnea



Dyspnea

on lying down

Cause:

Lt sided heart failure

Mechanism:

Pulmonary congestion

stimulation of j receptors.

Relieved by sitting position

* Lt sided heart failure

(endocrine) MAN P₂ ↑, H⁺, P_{O2} ↓

Some respiratory changes

Asphyxia

-- O_2 , $CO_2 \uparrow$, $H^+ \uparrow$

Causes

- 1 Air way obstruction.
- 2 Bilateral pneumothorax.
- 3 Closed system breathing.
- 4 Drowning.
- 5 Diaphragmatic paralysis.

Changes

1st ++ PCO_2 ++ H^+ -- PO_2
 ++ HR & ++ ABP.

++ Catecholamines.

then resp. stops. ①

ABP Falls.

Heart slows ②

Venous blood
has O_2

Apnea

Stop of breathing

Stoppage of respiration

due to inhibition of respiratory system.

Types of apnea:

- 1 Voluntary apnea.
- 2 Following voluntary hypervent.
- 3 Swallowing apnea.
- 4 Vomiting apnea.
- 5 Adrenaline apnea.
- ⑥ Apnea with Cheyne Stokes respir.
- ⑦ Sleep apnea

Periodic breathing

Physiological causes of periodic breathing:-

- 1 High altitude. Maxic
- 2 After voluntary hyperventilation.
- 3 In babies, during sleep especially premature babies.

Pathological states i.e. (Cheyne Stokes breathing):-

- 1 Respiratory failure e.g. brain damage or uremia

-- sensitivity to CO_2

- 2 Circulatory failure e.g. congestive heart failure.

Mechanism:

Respiratory centres are hypoxic $\rightarrow$ less responsive to CO_2 and H^+ $\rightarrow$ apnea.

Apnea $\rightarrow$ $\downarrow PO_2$ which stimulates the peripheral chemoreceptors $\rightarrow$ few breaths $\rightarrow$ $\uparrow PO_2$ $\rightarrow$ apnea.

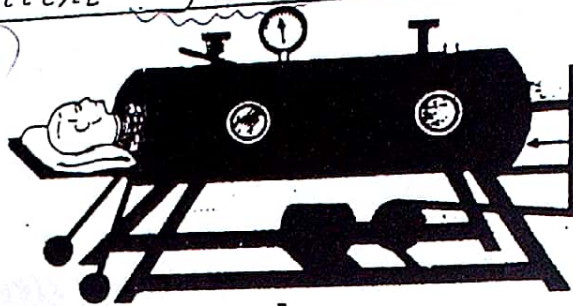
و تقييد الوجود في الوجود

Artificial respiration

- Intermittent positive pressure:
- Intermittent negative pressure:

acute resp. failure
chronic resp. failure

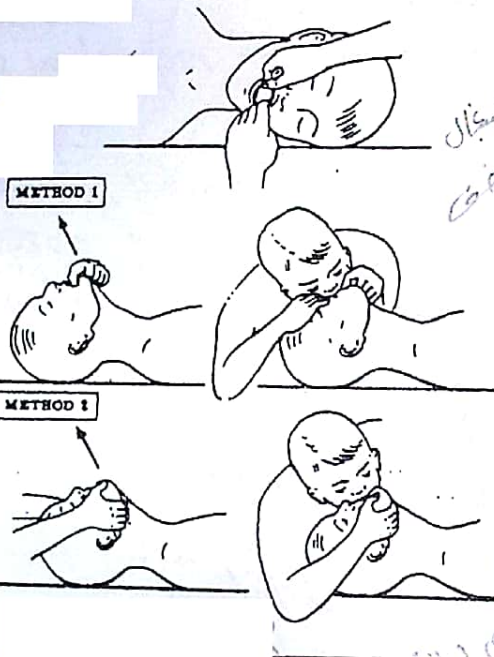
دكتور في الحيات
مدير في الحيات
(مدير في الحيات)



sleep 3 months in it
tank respirometer

3 Mouth to mouth:

emergency



القلب في الحيات
القلب في الحيات

tongue
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20 time / Min

Good Luck

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